VOLUME 22 July, 1956 NUMBER 7

THE AMERICAN SURGEON

PUBLISHED MONTHLY BY
THE WILLIAMS & WILKINS COMPANY
BALTIMORE 2, MARYLAND

Entered at the Post Office in Baltimore, Maryland as second-class matter.
Subscription price $10.00

CONTENTS, JULY

The American Surgeon

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Publisher

THE WILLIAMS & WILKINS CO.

Mt. Royal & Guilford Aves., Baltimore 2, Md.

THE
AMERICAN
SURGEON

Vol. 22, No. 7 July, 1956

DEFINITIVE RADICAL RESECTION OF THE LARGE BOWEL FOR RECURRENT DIVERTICULITIS*

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Diverticulosis is accepted as a term meaning the presence of uncomplicated diverticula in the intestinal tract, usually the sigmoid colon. Such diverticula are considered to be primary acquired single or multiple out-pouches or herniations of the mucous membrane through gaps in the muscularis of the colon. The end effect is that the mucosa lies directly under the serosa forming a small hernial pouch. Most of these diverticula are clinically unimportant. Their origin and mechanical factors have been amply discussed in medical literature.

It usually is assumed that diverticula are present in 5 to 10 per cent of all individuals over 40 years of age. Pemberton, Black and Maino reported an 8.5 per cent incidence found in a series of 47,000 barium enema examinations performed in the Mayo Clinic.

In a study of 2,000 consecutive barium examinations done in the Massachusetts General Hospital, it was found that there was a steady increase in the frequency of the disease as age progressed.

It is commonly taught that diverticulosis gives rise to no symptoms. This is not a sound precept since such a condition frequently produces mild symptoms such as a disordered action of the bowel; intermittent flatulence, and abdominal distention.

It has been estimated that about a fifth of the patients with diverticulosis develop clinical evidence of inflammatory reaction of such magnitude as to merit a diagnosis of diverticulitis.

If pain is a prominent feature, and especially if associated with positive physical signs, a diagnosis of diverticulitis and not of diverticulosis should be made. At times, it is impossible to determine by clinical and radiologic methods whether or not a mild inflammation is present. Comparatively, it presents similar difficulties of diagnosis to that of chronic appendicitis. When depending upon barium

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Presented during the Richmond Assembly of The Southeastern Surgical Congress, March 12-15, 1956, Richmond, Virginia.

enema roentgenologic examination to establish a diagnosis of diverticulitis, it is important to remember that from 5 to 10 per cent of sigmoid lesions will be missed by this type of study.

The majority of cases of diverticulitis are mild and as a rule, these patients with mild cases respond to a conservative form of treatment. However, some types of diverticulitis are not so mild in origin, and not all of them regress under a medical regime.

The occasional fulminating attack of diverticulitis occurs with severe abdominal pain, high fever, leukocytosis, and signs of spreading peritonitis. The patient with this type requires immediate, emergency surgical intervention.

Maingot has stated that 15 per cent of patients suffering from diverticulitis develop complications of such a serious nature as to warrant operative interference. Rankin placed the figure as 1 in 11 patients suffering from diverticulitis.

Modern revisions of textbooks as well as recent treatises on surgery of the gastrointestinal tract usually state "the primary treatment of diverticulosis and diverticulitis is medical, surgery being reserved for those subjects who have complications".

In an editorial statement in 1949, Lewis suggested that fixed routine methods of treatment are dangerous in diverticulitis, and that each case must be decided completely upon its individual merits.

In 1950, Boyden reported 25 primary resections without preliminary colostomy, and with no operative mortality. Since that time, ample evidence has been presented that primary resection in selected cases of diverticulitis is a safe procedure. This safety of surgical intervention has led to the idea that delay of operation until grave complications develop is no longer necessary, and that earlier definitive surgical attack on diverticulitis is wise.

In contrast to this changing concept, a revised edition of Maingot's Abdominal Surgery, published in 1955, states that the indications for operative treatment are the complications of diverticulitis, e. g., (1) acute diverticulitis with perforation and spreading peritonitis; (2) acute diverticulitis with localized abscess; (3) acute large bowel obstruction; (4) colovesical fistula; (5) diverticulitis complicated by carcinoma; and (6) diverticulitis associated with massive hemorrhage.

It is interesting that this concept of reserving surgical treatment for the severe types of diverticulitis has persisted. Prompt intervention when carcinoma is suspected is invariably advocated, even though clinically, a neoplastic lesion may be associated with any one or several of the complicating factors attributed to diverticulitis.

If surgical intervention is advocated under one circumstance, why should it be judicious to await complications under another circumstance? Although the basic lesion, diverticulitis, from the pathologic viewpoint is less threatening, nevertheless from the viewpoint of clinical progression and morbidity, it can be extremely catastrophic.

By awaiting one of the complications of diverticulitis to justify surgical intervention, patients are being exposed to the hazards of chronic recurring disability, devastating complications, multiple stage operations, and prolonged hospitalization.

In the past, the chief indications for surgery have been those of the complications of diverticulitis. A revision of this policy, which is now occurring, should be brought into sharper focus.

Since the advent of intestinal antisepsis, increasing attention has been paid to the possibility of reducing the morbidity and lessening the economic burden for the patient by definitive attack on the primary lesion.

Johns has stated "when referred for surgical treatment, the majority of patients with the complications of diverticulitis have already suffered long periods of disability, with much pain and loss of time from their occupation. Obviously, if these complications could be prevented, such patients would be greatly benefited."

It is suggested that patients with a clear cut diagnosis of uncomplicated diverticulitis, supported by a history of recurrent attacks and roentgenologic evidence, should be regarded as being suitable for operation after a second major attack. Establishment of such a diagmosis should be accepted as an important reason for sigmoid resection.

By selecting the time for surgical intervention, during a period of quiescence, the hazards of surgery during an acute inflammatory episode are avoided. In addition, the possibility of carcinoma, which must be entertained under such circumstances, is realistically approached.

Moore and associates, in reviewing the results of 22 patients coming to surgery because of sigmoid diverticulitis, stated that 12 patients were treated by sigmoid resection without benefit of preliminary colostomy. These authors stated that "it is believed that a general realization of the desirability for sigmoid resection at an earlier state in symptomatic diverticulitis would permit safe omission of preliminary colostomy in many cases".

Among the patients treated at the University Hospital, there were 10 in whom immediate surgery was palliative, and comprised either cecostomy or transverse colostomy. One patient with generalized peritonitis died 48 hours postoperatively. The remaining 9 patients, 2 of whom had colovesical fistulas, had definitive sigmoid resections on subsequent admissions. These 9 patients represented a total of 26 hospital admissions for a total period of 294 hospital days, or an average of 32.6 days per patient. This illustrates one of the penalties of delay in operating.

Cecostomy or colostomy should not be considered a definitive procedure for diverticulitis. Ultimately, the offending inflamed sigmoid should be resected.

Four patients were subjected to primary interval sigmoid resection without defunctionalization of the colon. These 4 patients represented 6 hospital admissions for a total of 76 hospital days, or an average of 19 days per patient.

One patient in this group, on his first admission, had a generalized purulent peritonitis with loculated abscesses. Peritoneal cultures revealed a moderately heavy growth of Escherichia coli and a light growth of Alpha streptococcus. A cecostomy was performed. This closed spontaneously. Approximately 3 months later an interval elective operation was done.

In addition to resecting 16 cm. of sigmoid colon, it was necessary to resect 30 cm. of ileum about 20 cm. proximal to the ileocecal valve, and to dissect inflamed

adherent sigmoid from the fundus of the bladder. The ileum and its mesenteric attachment were thickened, indurated and hemorrhagic.

Another patient in this group was admitted to the University Hospital complaining of abdominal pain and urgency and frequency of bowel movements. Her laboratory studies, proctoscopy and roentgenologic examinations, including barium enema, were entirely negative. Due to the persistence of her complaints, abdominal exploration was advised and accepted.

Exploration revealed a diffuse inflammatory mass in the sigmoid, infiltrating into the lateral pelvic peritoneum and the fundus of the bladder. There also was extensive lymphadenopathy in the mesentery and peritoneum. A primary sigmoid resection was done.

Despite the negative barium enema, lack of leukocytosis and inability to palpate a mass, subsequent pathologic report revealed induration and thickening of the bowel wall with multiple small abscesses. This type of case serves to illustrate the fact that severe pain, even when not associated with fever, leukocytosis and positive roentgenologic examination merits a diagnosis of diverticulitis rather than diverticulosis.

SUMMARY

It is suggested that a primary sigmoid resection is a safe procedure in uncomplicated diverticulitis. After a second major attack of diverticulitis, particularly when supported diagnostically by laboratory and roentgenologic evidence, an interval primary sigmoid resection and anastomosis is advocated.

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REFERENCES

1. Boyden, A. M.: Surgical treatment of diverticulitis of colon, Ann. Surg. *132:* 94 (July) 1950.
2. Glenn, F., and Harrison, C. S.: Surgical aspects of diverticulitis, Gastroenterology *20:* 643 (Sept.) 1953.
3. Johns, W. A.: Diverticulitis of colon-prevention of complications, Tr. South. S. A. *63:* 307, 1952.
4. Lewis, F. I.: Diverticulitis of colon, Ann. Surg. *130:* 983 (Nov.) 1949.
5. Maingot, R.: Abdominal Operations, 3rd. ed., New York, Appleton Century Crofts, Inc., 1955, p. 233.
6. Martin, J. D., Jr., and Caudle, R. S.: Diverticula of colon, South. Surg. *14:* 82 (Feb.) 1948.
7. Mayo, C. W., and Blunt, C. P.: Vesicosigmoidal fistulas complicating diverticulitis, Surg., Gynec. & Obst. *91:* 612 (Nov.) 1950.
8. Moore, R. M., and Kirksey, O. T., Jr.: One-stage resection in selected cases of sigmoid diverticulitis, Tr. South. S. A. *65:* 333, 1953.
9. Pemberton, J. deJ., Black, B. M., and Maino, C. R.: Progress in surgical management of diverticulitis of sigmoid colon, Surg., Gynec. & Obst. *85:* 523 (Oct.) 1947.
10. Rankin, F. W., and Brown, P. W.: Diverticulitis of colon, Surg., Gynec. & Obst. *50:* 836 (May) 1930.
11. Rankin, F. W., and Grimes, A. E.: Surgical treatment of diverticulitis, South. Surg. *7:* 1 (Feb.) 1938.
12. Ransom, H. K.: Diverticulitis of colon, Gastroenterology *26:* 12 (Jan.) 1954.
13. Stone, H. B.: Surgery of colon, Northwest Med., *41:* 412 (Dec.) 1942.
14. Idem: Surgical aspects of certain non-malignant colon conditions; chronic nonspecific ulcerative colitis, diverticulitis, megacolon, Virginia M. Month. *78:* 3 (Jan.) 1951.
15. Welch, C. E., Allen, A. W., and Donaldson, G. A.: Appraisal of resection of colon for diverticulitis of sigmoid, Tr. Am. Surg. A. *71:* 44, 1953.

ADVANCES IN TECHNICAL ASPECTS OF BILIARY TRACT SURGERY

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Presentations on technical aspects of surgery have been infrequent on programs in the last two decades. The reasons for this are several. One undoubtedly is because significant scientific advances justifiably have had emphasis and reemphasis to disseminate the knowledge which makes surgery safer. Most of these of course have to do with preoperative and postoperative care. Another reason why technical papers have not been popular is that they undoubtedly can draw an unfavorable reaction from the audience who may think that because the author presents these details vocally he must regard himself very proudly. After all, it is still true that a patient being operated upon is in the hands of the surgeon and the outcome depends entirely during this phase of the management upon him. There is something mechanical about it, something that can be highly exact and skillful and on the other hand bungling and disastrous. It is still true that each of us would pick a surgeon whom we think is technically superior to operate upon us, and it will remain that way in spite of all the other refinements and advances. Judgment and execution of the technical aspects remain the most important single feature in a surgical case. If they were not, the clinicians could compete handsomely with us in our own field.

One of the first fundamentals in operations for benign lesions of the biliary tract is to have good anesthesia and to have an adequate approach and exposure. The choice of anesthetic agent is a digression which permits a lot of justifiable discussion. There is no one incision for the approach to the gallbladder or common bile duct. There are a number and there is a choice. I prefer the Mayo-Robson incision and where there is a stricture of the biliary tract in the hilum of the liver a thoraco-abdominal incision.

EXPLORATION OF THE ABDOMEN

It is well for surgeons to recall at the time of the first operation for a biliary tract lesion that there is such a thing as postcholecystectomy syndrome. The incidence of this can be reduced by carefully checking all the possibilities which could contribute to symptoms and in each operation there must be a routine firmly adhered to, so that nothing can be overlooked in spite of the fact that the biliary tract lesion is evident. Any lesion or condition which is possibly a contributory factor in the symptoms must not be disregarded or undiscovered. For that reason it is well each time to explore a patient in the same routine fashion. The direction should not be varied for it is necessary to check these organs and

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Presented during the Richmond Assembly of The Southeastern Surgical Congress, March 12–15, 1956, Richmond, Virginia.

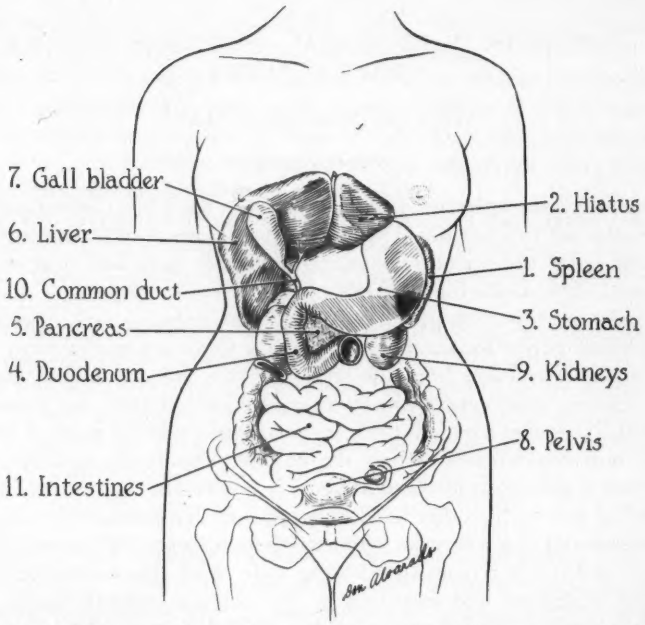

FIG. 1. A satisfactory routine order for exploring the abdomen at the beginning of operations for benign lesions of the biliary tract. The spleen should be checked, then the esophageal hiatus, then the stomach, the duodenum, the pancreas, the liver, the gallbladder, the pelvis, the kidneys, the common duct, and if indicated, the entire small intestine. If this or an analogous type of routine is strictly followed the surgeon is not apt to overlook lesions which may be contributing to the symptoms and which subsequently may be responsible for a postcholecystectomy syndrome.

conditions and a routine will stimulate accuracy. At first the spleen is checked for any enlargement or perisplenitis and then the esophageal hiatus for a hernia and then the stomach for a mass or evidence of an ulcer and then the duodenum, not only the first portion but the descending portion, particularly with thought of ulcer or diverticulum. Then the pancreas is palpated for size and for consistency, noting if there is any difference in consistency in the head or the tail or any enlargement throughout the organ or in a specific part. Then attention is turned for the first time to the liver and then the gallbladder. It is important to check the superior surface of the liver. I have encountered a small amoebic hepatic abscess and neoplasms which were undiagnosed preoperatively by this careful examination. The next structure to evaluate is the common duct. Now the common duct and the cystic duct usually cannot be seen in the gastrohepatic omentum but numerous illustrations show them as shining clearly through as if they were perfectly evident. They are not. They are evident only later after the surgeon exposes them. At this stage the gastrohepatic omentum is palpated, carefully feeling for stones or masses or induration. Then the kidneys are checked and it is well to remember the adrenals capping the superior pole of each kidney

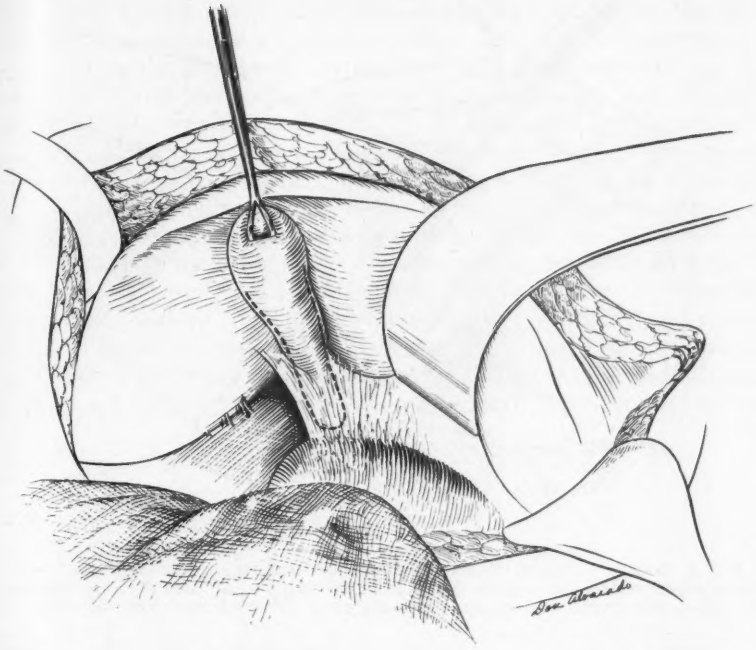

FIG. 2. At the beginning of the biliary tract operation one is faced with obscurity of anatomic detail. Particularly in the obese patient the ducts are not visible and it is absolutely necessary to see them (and clearly); the common duct above and below the entrance of the cystic duct and the cystic duct in its entirety.

at the time one is feeling and thinking of a possibility of a lesion. Then the pelvis is checked, particularly in the female, and some attention should be paid to the possibility of induration in the sigmoid or a mass there. I usually leave the evaluation of the appendix and cecum to the termination of the operation. As a general rule it is not essential to inspect every inch of the small intestine but if it is justified on the preoperative suspicions, this may be added to the exploratory phase of the operation.

When all this is done attention is turned to progress in the technical part of the biliary tract operation. Usually the liver is brought down after the suction affect of its space against the diaphragm has been liberated by inserting the hand over the dome.

CHOLECYSTECTOMY

Certain technical features in the removal of a gallbladder are extremely important. If it is indicated to remove the gallbladder it may be clamped and retracted upward in the wound. Whether one chooses to remove a gallbladder from ampulla to fundus or the reverse makes little difference. It may be removed either way. The most essential feature in cholecystectomy is to expose the common

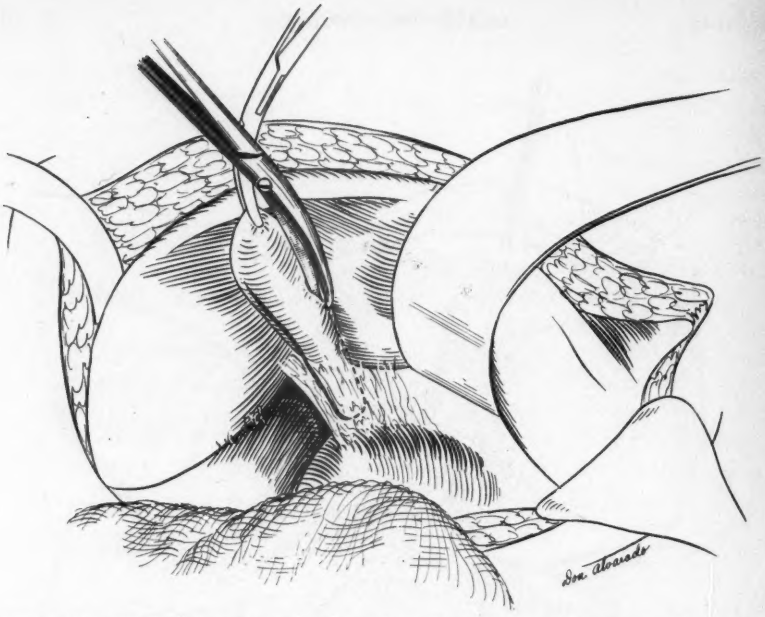

FIG. 3. In order to follow the principle of clear visualization it is necessary to cut carefully the peritoneum and to push it back from the ampulla and from the anterior aspect of the gastrocolic omentum overlying the junction of the cystic and common ducts.

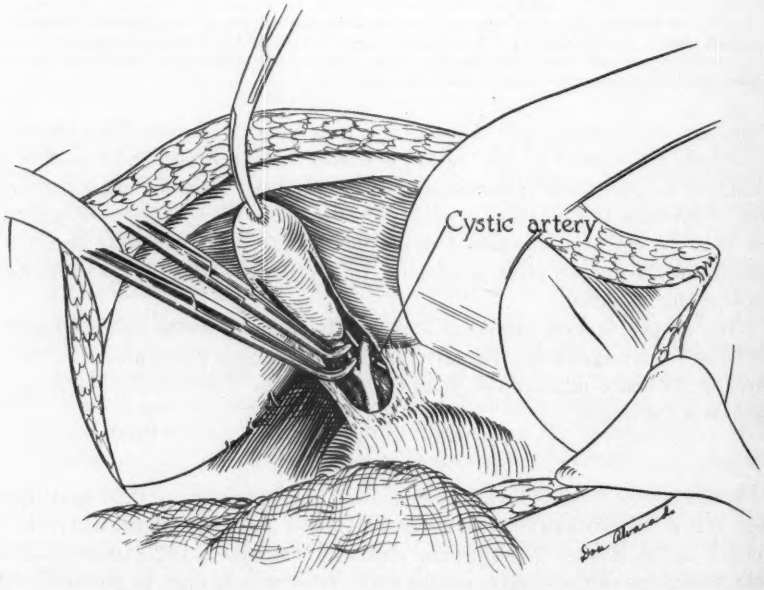

FIG. 4. After clear visualization is established the cystic artery and cystic duct are evident and technical errors will not occur.

bile duct and the cystic duct and to show clearly the common bile duct above and below the entrance of a cystic duct into it. This cannot be accomplished without cutting the peritoneum. So the first step in actual removal of the gallbladder is to cut the peritoneum on one side of the gallbladder about 1 cm. away from the hepatic edge. The peritoneum on the gallbladder is incised carefully running parallel to the liver down around the ampulla and down over the common duct. Another incision on the peritoneum is made on the other side of the gallbladder and 1 cm. away and parallel to the liver running down around the ampulla and the cystic duct, joining over the common duct the previous incision. This careful cut in the peritoneal attachment of the gallbladder to the liver is analogous to the better known incision in the peritoneum at the lower segment of the uterus anteriorly to reflect the bladder when performing hysterectomy. With a careful dissector the peritoneum then is pushed back from the cystic and common ducts. The entire cystic duct comes into clear view and the common duct above and below the cystic duct becomes evident. Then one sees how large

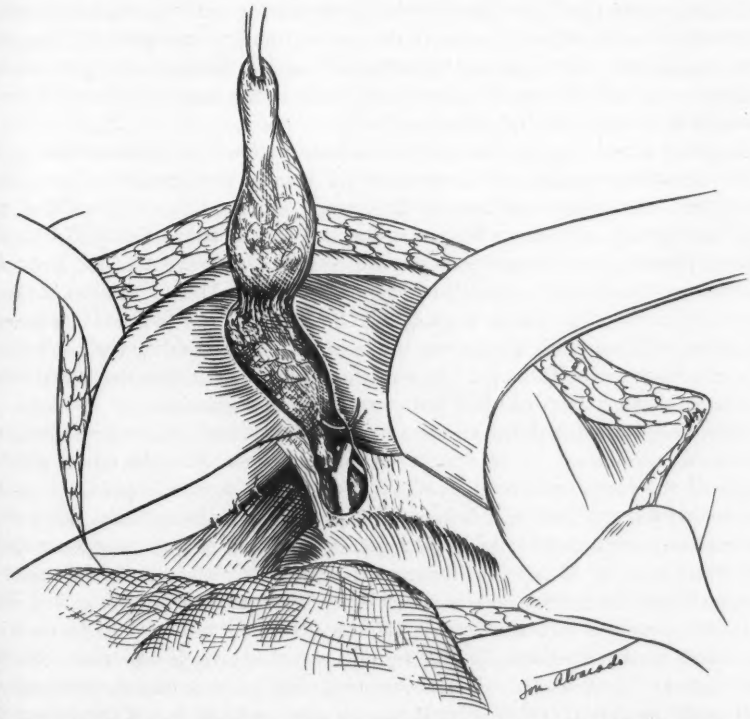

FIG. 5. After clamping and severing the cystic duct in close proximity to the common duct accurate ligatures may be applied and the gallbladder is removed from its bed leaving loose fibroareolar tissue as a capsule on the liver. If feasible it is better to suture the marginal peritoneum over the gallbladder bed.

the common duct is; also sees any induration or thickening in its wall. During this phase of dissection the cystic artery comes into view. Sometimes a large right hepatic artery has a very short cystic stem coming off in intimate contact with the ampulla of the gallbladder and the cystic duct. This important artery may be injured by failure to properly visualize the cystic duct before clamps are applied.

I usually remove the gallbladder from its bed from ampulla to fundus. If this is the choice, Moynihan forceps may be applied to the cystic duct in close proximity to the common duct under clear visualization, and the gallbladder is removed leaving fibroareolar tissue capsule attached to the liver.

EXPLORATION OF THE COMMON BILE DUCT

When indications for exploring the common bile duct are definite the method of exploring it with greatest safety and thoroughness is extremely important. Perhaps a cholangiogram at the time of operation will prove of value. At present it has its pitfalls and inaccuracies but these are being obviated, and ultimately cholangiography may be more reliable. If cholangiography shows true or ghost shadows simulating stones one does more to try to find and remove them. Shouldn't it be a philosophy for surgeons that they do such a thorough job prior to cholangiography that the necessity for going back at the same or subsequent operations will be reduced to a minimum?

In order to accomplish this certain conditions must be present and among those deserving mention are instruments. I believe instruments are extremely important. Consider stone forceps. If they are too tight or too loose they may mar the delicacy and accuracy of the surgeon's touch. If forceps are stiff a surgeon cannot possibly be accurate and precise. Another instrument which justifiably may be mentioned are common duct sounds or dilators. Most of the Bakês variety have 7½ in. handles. These are too short for many obese patients. I devised a Bakês type dilator with a groove in the head to assist in guiding the knife during ampullotomy and with long 11 in. handles. The surgeon inserting these or the assistant holding them will find the extra length a decided help.

Combined supraduodenal and transduodenal approach is being adopted with increasing confidence. In 336 operations for benign lesions of the biliary tract we explored the common bile duct in 26.5 per cent. Combined supraduodenal and transduodenal exploration was used in 53 per cent and the supraduodenal choledochostomy without transduodenal approach in 47 per cent. Although we opened the duodenum in 47 patients during exploration of the common bile duct for benign biliary tract lesions we have not had a hospital death in this group. There were two deaths in 89 explorations of the common duct but the two deaths which occurred, occurred when only the supraduodenal approach was used. This does not indicate that combined transduodenal and supraduodenal exploration is not more dangerous technically. It has an increased risk but it does mean that it can be done relatively safely. There is an advantage in opening the duodenum in certain cases. This advantage may outweigh the minor additional risk entailed in doing it.

The indications I use for opening the duodenum to have additional access to the common bile duct are these:

1. When there are stones in the common bile duct.
2. When there is evidence of pancreatitis or hard or enlarged pancreas.
3. When there is any doubt about the transition of the dilator into the duodenum through the papilla of Vater.
4. When it is a secondary operation for biliary dyskinesia or postcholecystectomy syndrome.

The first step in exploring the common bile duct is made when the common and cystic ducts are clearly exposed and the gallbladder still remains in its position. There is some advantage in exploring the common duct prior to removal of the gallbladder. It may be used for traction to put the common duct on extension and expose the space between the cystic duct and duodenum. Better visualization also may be obtained by liberating the upper half of the duodenum from its right lateral peritoneal attachment. The incision to liberate the duodenum is carried from its first portion at the gastrohepatic omentum down the right lateral border posteriorly and then the surgeon may use his fingers to gently elevate the duodenum lifting it forward together with the head of the pancreas. It is notable that the posterior aspect of the duodenum is covered with peritoneum even beyond the severed peritoneal reflection. This fact is not widely known since it is stated that the duodenum is a retroperitoneal organ which might be interpreted to mean that the posterior part is not covered by peritoneum; but it is. The only portion of the duodenum not having a serosa is the juxtapancreatic portion. The longitudinal incision having been made in the common duct about ½ cm. in length and below the cystic duct, scoops, probes, dilators, forceps and irrigators may be introduced. If a Bakês dilator is introduced it may give the impression that an obstruction exists or that it passes freely into the duodenum but it is possible to mistake one's judgment as to the transition of a dilator into the duodenum through the papilla of Vater. The ampulla of Vater is remarkably free and unattached and it may be displaced widely without permitting the dilator to transverse it. It may be pushed up against the other wall with the dilator giving the impression that it is in the duodenum when in reality it is simply pushing the papilla of Vater before it.

If a transduodenal approach also is decided upon the superior leaf of the transverse mesocolon must be incised and pushed down off the anterior aspect of the duodenum since it crosses the upper half of the second portion. The transverse mesocolon is surprisingly high on the duodenum and obscures the entire lower half of its descending portion. It may be incised and pushed down so that the second and third portion of the duodenum are clearly evident.

If the duodenum is opened it should be opened transversely and preferably at the spot which is indicated by a Bakês dilator. In this a long-arm dilator is especially helpful. Only a small incision is necessary. Why a transverse instead of a longitudinal incision? Because the incision is made on the greater curvature of the duodenum. Tension is undesirable, particularly if there is any evidence of pancreatitis or edema in the region. If a longitudinal incision is made and it is

closed transversely the greater curvature is shortened and there is more apt to be tension than if one closes the transverse incision transversely. There need not be much turn-in and the lumen is not compromised to any extent. Usually when transduodenal exploration is indicated it may be advantageous to do an ampullotomy. I prefer to do it on a Bakês dilator with a specially provided slot for cutting the papilla with a knife. I do not think there is necessity to suture the mucous membrane of the duodenum to the common duct at the edges of the ampullotomy but have no contentious remarks with regard to those who prefer to do it. When the ampulla is cut I usually leave a long-arm T tube transvaterianally into the duodenum and remove it only after a 4 to 6 week period. This is with the idea of providing an adequate opening which does not stenose in healing. In order to prevent obstruction of pancreatic and biliary drainage by the T tube passing through the ampulla, one must put multiple openings in the vertical limb. In this way, no pancreatitis develops.

It is not necessary to resuture the peritoneum over the common duct and at the posterior aspect of the duodenum. After adequate drains have been placed the abdominal wound is closed.

SUMMARY

A discussion of certain important technical features in operations for benign lesions of the biliary tract is presented, emphasizing technical safeguards in removing the diseased gallbladder and the details of common duct exploration, supraduodenal only and combined supraduodenal and transduodenal.

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THE DIAGNOSIS OF PERFORATING ARTERIOSCLEROTIC ABDOMINAL AORTIC ANEURYSMS: A REVIEW OF CASES

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It now has been established that perforated or leaking arteriosclerotic abdominal aortic aneurysms can be treated successfully by resection and replacement grafts. The emergency nature of the procedure and urgency of making the diagnosis early is obvious.

As the term implies, this aneurysm develops as a result of arteriosclerotic degeneration in the vessel wall. The arteriosclerotic abdominal aortic aneurysm occurs in older aged patients; it is fusiform, it arises below the renal arteries, and rarely produces erosion of the vertebrae, whereas, the syphilitic abdominal aortic aneurysm is saccular, arises in the upper abdominal aorta above the renals, frequently produces erosion of the vertebrae, and occurs in middle aged patients. The arteriosclerotic abdominal aortic aneurysm frequently presents as a pulsatile, midabdominal mass without producing any distressing symptoms. The walls vary in thickness from paper thin areas to areas which have become quite thick as a result of laminated clot formation. It is not to be confused with dissecting aortic aneurysms, in which case, as a result of medial necrosis, there is a split in the intima and media of the proximal aorta with a spreading intramural hematoma developing, sometimes throughout the entire length of the aorta from the arch distally.

Prior to resection of the aneurysm and graft replacement of the abdominal aorta, there was no satisfactory treatment for the condition. Milton[14] was the first surgeon to operate for a ruptured abdominal aortic aneurysm when he ligated the aorta proximal to the aneurysm. The patient died 24 hours later.

Using the principle of wiring, introduced by Moore,[15] and improvising electrothermic coagulating current to further obliterate the aneurysm, Blakemore[2] has reported temporary improvement in the treatment of certain abdominal aortic aneurysms, some of which had perforated. Since Lowenberg[13] recommended wrapping this aneurysm with skin, various materials have been used, hoping to induce fibrosis of the walls. Waugh[11] used polyvinyl sponge to wrap a leaking abdominal aortic aneurysm.

The resection of arteriosclerotic aneurysms of the abdominal aorta and replacement by preserved aorta was first reported by Dubost and associates,[5] and Schafer and Hardin.[16] In 1953, using this method, it was demonstrated by Bahnson[1] that leaking abdominal aortic aneurysms could be successfully treated.

With this background, it is obvious that little effort was made by the clinician to make a diagnosis when the ultimate outlook was so hopeless. With the ready availability of aortic homografts in certain medical centers, and the use of fabric prosthesis as replacement grafts, the emergency surgical treatment of abdominal

* From the Baptist Memorial Hospital and Sanders Clinic, Memphis, Tennessee.

aortic aneurysms has become feasible and successful treatment has been reported by various surgeons.[1, 3, 8, 9]

Kirklin[12] used nylon prosthesis as replacement graft following resection of a perforated abdominal aortic aneurysm.

NATURAL HISTORY OF PERFORATING ABDOMINAL AORTIC ANEURYSMS

Estes,[6] in a study of abdominal aortic aneurysms at the Mayo Clinic, reported 6 cases in which the patients symptoms were considered the result of rupture of the aneurysm. One patient died the day of onset, 2 patients lived 5 days, and 2 patients survived 4 weeks. One patient underwent surgical exploration for symptoms. An abdominal aneurysm with a large retroperitoneal hematoma was found, but repair was not attempted. She then lived 4 years before dying of a ruptured aneurysm.

Blakemore[2] states there is almost invariably an interval of from 2 to 6 days following the first retroperitoneal hemorrhage before death supervenes from sudden rupture of the retroperitoneal hematoma into the peritoneal cavity. It is of interest to note that of 84 arteriosclerotic abdominal aortic aneurysms which were operated upon by Debakey and associates,[4] 17 were perforated.

The purpose of this study is to focus attention on certain diagnostic features of this emergency condition so there may be minimal delay instituting surgery.

The following cases are from the files of the Baptist Memorial Hospital and Sanders Clinic, Memphis, Tennessee, during the years from 1949 through 1954.

CASE REPORTS

Case #1. F. H. G., a white man, aged 82, was admitted to Baptist Memorial Hospital in 1952 with a history of the sudden onset of severe, sharp, right lower quadrant abdominal pain of 2 hours' duration. The pain was not radiating; there was no nausea or vomiting. He was cold, clammy, and cyanotic. The blood pressure was 65/0, pulse 88. The heart sounds were faint, the abdomen slightly distended, with a tender pulsating mass in the mid and right lower quadrants of the abdomen. The white blood cell count was 18,000 per cu. mm.

The patient's history revealed an abdominal aortic aneurysm had been demonstrated one year prior to this admission.

He responded to blood transfusions and vasopressor drugs, and 3 hours after admission was explored with the diagnoses of dissecting aneurysm, mesenteric thrombosis, and acute intesintal obstruction considered.

At operation, a pulsating mass in the retroperitoneal area was found. This mass was approximately 8 cm. in diameter and was located in the midline with a large retroperitoneal hematoma on the right side. Nothing was done, and the abdomen was closed. The patient died 4 hours later.

The autopsy revealed a retroperitoneal hematoma behind the ascending colon (estimated 2–3 liters). Also present was an aneurysm of the lower abdominal aorta 10–6 cm. with perforation of the right lateral border.

Case #2. C. P., a white man, aged 63, was admitted to the hospital with acute left flank pain of 1 hour's duration, which pain was so severe it required opiates for relief.

Physical examination on admission revealed a slight abdominal distention with a questionable mass in the midabdomen just to the left of the umbilicus. The blood pressure was 174/100 and his pulse was 86. The white blood cell count was 18,150 per cu. mm., the red blood cell count was 3,610 per cu. mm., hemoglobin was 9.8 Gm. Scout film of the abdomen

revealed a curvilinear distribution of calcium streaks to the left of the midline in the lumbar area with the suggestion of a retroperitoneal mass in the left side.

This patient had a history of hypertensive arteriosclerotic vascular disease.

Four hours after admission, he became sweaty, pale, and the pulse became thready; his blood pressure dropped to 90/70. Repeated blood transfusions and vasopressor drugs were given with only temporary response, and the patient died 15 hours after admission.

The final clinical diagnosis was ruptured arteriosclerotic aneurysm of the abdominal aorta. No postmortem examination was made.

Case #3. E. S. D., a white man, aged 66, was admitted to the hospital because of severe pain in the right costovertebral angle and right flank, and no bowel movements for 4 days.

This patient had had right flank pain for 1 month, and had vague urinary disturbances with urinary retention on one occasion. He was an opiate addict.

On examination, there was tenderness in the right flank and right inguinal region. The abdomen was distended, and peristalsis increased. The blood pressure was 136/98, pulse 90, white blood cell count was 24,500 per cu. mm., red blood cell count was 3,260,000 per cu. mm., hemoglobin was 10.8 Gm., and the serology was negative for syphilis. Possible diagnoses were obstructive lesion of the colon or right renal disease.

A scout film of the abdomen reported the colon and small bowel were filled with gas with the suggestion of a mass in the midabdomen, not typical of intestinal obstruction.

A repeat examination 5 hours after admission showed evidence of a right abdominal mass. Within the hour the patient suddenly became pale and weak, went into profound shock, and died 6 hours after admission to the hospital.

The final clinical diagnosis was abdominal aortic aneurysm with rupture. No autopsy was granted.

Case #4. V. T. A., a white man aged 75, was admitted to the hospital with excruciating upper abdominal pain which radiated to the low lumbar back area, and had been present for 20 hours. He was nauseated and vomited, with some bright red blood in the vomitus. Four hours previous to hospitalization, his blood pressure dropped to shock level and he became comatose.

The patient had a history of hypertension for several years. He had had a stroke about 4 years before, from which he had almost recovered.

This man was unconscious, his blood pressure was 40/0, temperature 96 F., and the peripheral pulse unobtainable. Abdominal examination revealed an 8 cm., barely pulsatile mass, just to the left of the umbilicus. The white blood cell count was 13,350 per cu. mm., red blood cell count was 3,350,000 per cu. mm., hemoglobin was 8.5 Gm. Urinalysis showed 1 plus albumin and with occasional cast. The Kline test was negative.

The diagnosis was arteriosclerotic aneurysm of the abdominal aorta with slow leakage.

He was given vasostimulants, blood transfusions with temporary elevation of blood pressure, but it did not become stabilized. The patient remained in a coma, was oliguric, and died 24 hours after admission. No autopsy was done.

Case #5. J. T. F., a white man, aged 62, was admitted to the hospital with a severe low back pain, radiating to the testicles, of 2 weeks' duration. He had noted an abdominal mass for several months which was gradually increasing in size.

This man had had hypertension for several years, and a diagnosis of abdominal aortic aneurysm had been made elsewhere 2 weeks prior to this admission. The white blood cell count was 15,000 per cu. mm. The Kline test was negative.

Two days after admission, he suddenly went into a state of shock, with the simultaneous development of a mass in the left flank. The blood pressure was 80/50, peripheral pulses were thready, and there was no left femoral pulse.

He responded to multiple transfusions, and the next day his blood pressure was 190/120. He appeared to be improving when he suddenly developed shock 4 days after admission and died.

The autopsy revealed he had a 14 cm. abdominal aneurysm which extended down into

both iliacs with rupture retroperitoneally, and a large retroperitoneal hematoma. No clear cut site of the rupture was noted.

Case #6. Mrs. F. F., a white woman, aged 79, was admitted to the hospital as an emergency. She complained of lower abdominal pain, nausea and vomiting, of 5 days' duration. She had vomited bright blood on one occasion.

On physical examination, she appeared to be in mild shock with blood pressure of 100/78, pulse 110, and temperature 99.8 F. There was slight abdominal distention; bowel sounds were present. There was a pulsating mass in the lower abdomen to the left of the midline.

Laboratory studies revealed the red blood cell count was 2,279,000 per cu. mm., white blood cell count was 12,600 per cu. mm., the nonprotein nitrogen was 54 mg. per cent.

The tentative diagnosis was obstructive lesion of the left colon with perforation and peritonitis.

She was given blood transfusions and responded very satisfactorily. Gastric suction siphonage also was started, and the following day there was less abdominal rigidity and normal bowel sounds. Peritonitis also appeared less evident. Intravenous glucose solution and blood were given during the next 24 hour period.

About 34 hours after admission, the patient again developed shock and died 36 hours after admission.

Necropsy revealed a perforated, 6 cm. aneurysm of the abdominal aorta about 4 cm. below the renals. There was a retroperitoneal and intraperitoneal hematoma of approximately 1500 cc. volume. The aorta was sclerotic, and there was generalized arteriosclerosis and cardiac hypertrophy. No definite site of the perforation was noted.

Case #7. W. G. L., a white man, aged 73, was admitted to the hospital complaining of left lower abdominal pain of 2 hours' duration, and a history of transitory *"black out"*, loss of consciousness at the onset. He also had the desire to defecate with the onset of the abdominal pain. He had had no symptoms prior to the onset of the present illness.

Examination revealed he was in mild shock, and was semiconscious. His blood pressure was 74/50, pulse 94. Femoral pulses were present. There was slight abdominal distention, tenderness, and splinting in the left abdomen with the suggestion of a left midabdominal mass. The laboratory findings revealed the red blood cell count was 2,910,000 per cu. mm., white blood cell count was 14,000 per cu. mm., and serology was negative.

The tentative diagnosis was perforated diverticulitis of the left colon with peritonitis and mesenteric thrombosis.

The treatment consisted of blood transfusion and intravenous glucose solution. The patient developed excruciating pain in the left hip, groin, and thigh. The following day ecchymosis of the scrotum was noted.

He suddenly died approximately 48 hours after admission. The necropsy report showed ruptured arteriosclerotic aneurysm of the lower abdominal aorta with large retroperitoneal hematoma. There was a 2 cm. defect on the anterior wall of the aneurysm communicating with the hematoma.

Case #8. L. R. L., a white man, aged 59, was admitted to the hospital in mild shock, with the history of the sudden onset of severe abdominal pain with collapse 24 hours previously. He was admitted to the hospital in his home town where he received blood transfusions and vasostimulants. A diagnosis was made there of leaking abdominal aortic aneurysm, and he was transferred by ambulance to this hospital, which is a distance of about 80 miles.

He gave a history of abdominal discomfort of 2 months' duration.

Physical examination revealed a very obese man in mild shock, with a pulsatile abdominal mass. The right femoral pulse was absent, and the left femoral pulse was questionable. Admission laboratory studies were as follows: red blood cell count was 2,600,000 per cu. mm., hemoglobin was 8.5 Gm., white blood cell count was 2,600,000 per cu. mm., packed cell volume was 27.5 vol. per cent, urinalysis was 1 plus protein.

Repeated blood transfusions were given during the next 24 hours along with vasostimu-

lants, but the patient remained in a state of mild to severe shock, and died 27 hours after the present hospital admission. During this time he vomited 75 cc. of dark blood stained musus.

Case #9. C. H. H., a white man, aged 68, was admitted to the hospital with a history of the onset of lower abdominal pain and cramping of 10 days' duration. There was some nausea and daily vomiting. The patient was an alcoholic addict, and was able to take little by mouth other than a liberal amount of whiskey daily.

He had a history of hypertension of several years' duration, and loss of weight from 262 pounds to 195.

Physical examination revealed an acutely ill, obese man, rather dehydrated. The abdomen was slightly distended. There was a tender mass in the right lower adomen with slight rigidity. His blood pressure was 170/70.

The laboratory studies on admission showed red blood cell count was 4,680,000 per cu. mm., hemoglobin was 73 per cent, white blood cell count was 15,750 per cu. mm., urinalysis was negative, the nonprotein nitrogen was 48.

Scout film of the abdomen reported as showing an over-all haziness.

The patient was explored through a McBurney incision, and a large retroperitoneal hematoma in the right side was noted. Nothing was done. The patient recovered, and left the hospital after 10 days. The blood pressure was 210/96 on dismissal. He was lost track of, and no follow up was made.

The final diagnosis was arteriosclerotic hypertensive disease with possible perforation of the abdominal aneurysm.

DISCUSSION

The diagnosis in 5 of the cases was proved either by necropsy or abdominal exploration. In 4 patients, the history, clinical findings, and hospital course were considered characteristic of perforated arteriosclerotic abdominal aortic aneurysms.

There were 8 men and 1 woman; the youngest patient was 59 years of age, the oldest patient was 82. The past history revealed many of the patients had had symptoms for several weeks, or even months, which could be attributed to expanding abdominal aortic aneurysms.

The presenting symptoms were usually those of an acute intra-abdominal condition. There was a history of fainting or collapse at onset, and all patients presented shock, varying from mild to profound. The abdominal pain and cramping was of a colicky nature, and frequently there was associated nausea and vomiting. In most patients, there was associated back or lumbar pain which radiated to the hips or inguinal region. Ecchymosis of scrotum was noted in 1 patient. A previous diagnosis of abdominal aortic aneurysm had been made in 2 cases. Such conditions as acute intestinal obstruction, mesenteric thrombosis, diverticulitis of the colon with peritonitis, acute appendicitis, cholecystitis with perforation and peritonitis, acute pancreatitis, and acute renal disease were considered in the differential diagnosis.

There have been previous reports wherein this condition has been mistaken for an acute intra-abdominal condition. Karabim[10] reported the case of a patient who was operated upon for acute appendicitis in whom he found a large retroperitoneal hematoma arising from a ruptured aortic aneurysm. Fernbach and associates,[7] reported finding a large retroperitoneal hematoma arising from a perforated iliac aneurysm, in a patient who was explored for acute appendicitis.

There was a certain amount of abdominal distention in all patients, and in 8 of the 9, an abdominal mass was noted. This was described as pulsatile in 5 patients. Ecchymosis of the scrotum was noted in one patient.

Abdominal rigidity was not remarkable in any patient, certainly not as marked as seen in acute peritonitis resulting from a free perforation of a viscus. Usually there was abdominal tenderness, particularly over the mass. Peristalsis usually was present in most instances, although decreased in some.

Laboratory studies revealed a leukocytosis in all patients, usually the white blood cell count was above 12,000 per cu. mm. The hemoglobin determination and red blood cell count were decreased in all patients, and all responded to blood transfusions. The serology was negative in all cases in which it was reported.

Roentgen examination of the abdomen, consisting of anterior posterior scout film was not particularly helpful. In most patients, there was noted small bowel dilatation, generalized abdominal haziness, and loss of psoas shadow in some. In one patient, an area of crescent shaped calcification, interpreted as the wall of an aneurysm, was described.

It is of interest to note that lateral roentgenogram was not made in any patient. Such an examination might be most helpful in demonstrating the presence of a retroperitoneal hematoma or a dilated calcified aorta.

A pulsatile abdominal mass was noted in 5 patients. Since pulsation in the mass would depend, to a certain extent, on the degree of shock at the time of examination, and to a lesser extent on the magnitude of the retroperitoneal hematoma, a nonpulsatile mass does not rule out perforated abdominal aortic aneurysm. The presence of femoral pulsations does not rule out perforated abdominal aortic aneurysm, although they may be weak or even absent. The interval from onset of the acute episode until abdominal exploration or death ranged from 9 hours to 14 days.

SUMMARY

From this study, certain characteristics and differential diagnostic points pertaining to perforated arteriosclerotic abdominal aortic aneurysms are noted.

All patients were over 58 years of age; males were predominant, the ratio being 8 males to 1 female. In 7 patients, there were past histories of hypertensive cardiovascular disease. A history of vague abdominal pain or lumbar pain radiating to the hip, inguinal region, or scrotum, antedating the acute episode of perforation for several days or weeks is not uncommon. Fainting or collapse with the onset of the acute episode, and the fact that all patients responded temporarily to blood transfusions is significant.

Physical examination revealed mild to profound shock, an abdominal mass which was often pulsatile, slight abdominal distention, normal or diminished peristalsis, tenderness on deep palpation over the mass, and only slight abdominal rigidity.

The white blood cell count was elevated in all patients, ranging from 12,000 to 24,500 per cu. mm. The hemoglobin and red blood cell count were diminished in all patients. Blood serology for syphilis was negative.

Roentgenologic studies, scout film of the abdomen, antero-posterior view, revealed abdominal haziness, a suggestion of an abdominal mass, slightly dilated loops of small intestine, and in 1 patient, a curvilinear calcific shadow, representing the calcified wall of the aneurysm.

There is a latent period, in most patients, from the time of original perforation of the aneurysm until fatal rupture ensues, and so called expectant treatment or delay in making the diagnosis may cost the patient his only chance for recovery. As soon as the condition is suspected, efforts are made to prepare the patient for immediate surgery.

With a greater awareness of this condition, resection of the aneurysm and graft can be done earlier, and the salvage rate of otherwise hopeless patients will be improved.

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REFERENCES

1. Bahnson, H. T.: Treatment of abdominal aortic aneurysm by excision and replacement by homograft, Circulation *9:* 494 (April) 1954.
2. Blakemore, A. H.: Progressive constrictive occlusion of abdominal aorta with wiring and electrothermic coagulation, Ann. Surg. *133:* 447 (April) 1951.
3. Cooley, D. A., and DeBakey, M. E.: Ruptured aneurysms of abdominal aorta (excision and homograft replacement), Postgrad. Med. *16:* 334 (Oct.) 1954.
4. DeBakey, M. E., Cooley, D. A., and Creech, O., Jr.: Aneurysms and occlusive diseases of aorta, Henry Ford Hospital, International Symposium on Cardiovascular Surgery, Philadelphia, W. B. Saunders Co., 1955, p. 469.
5. Dubost, C., Allary, M., and Oeconomos, N.: Resection of aneurysm of abdominal aorta, A.M.A. Arch. Surg. *64:* 405 (March) 1952.
6. Estes, J. E.: Abdominal aortic aneurysms: study of 102 cases, Circulation *2:* 258 (Aug.) 1950.
7. Fernback, P. A., Berman, L., and Cohen, B.: Intraperitoneal rupture of iliac aneurysm with survival following surgical treatment, Ann. Surg. *135:* 570 (April) 1952.
8. Gerbode, F.: Ruptured aortic aneurysm, surgical emergency, Surg., Gynec. & Obst. *98:* 759 (Dec.) 1954.
9. Javid, H., Dye, W. S., Grove, W. J., and Julian, O. C.: Resection of ruptured aneurysm of abdominal aorta, Ann. Surg. *142:* 613 (Oct.) 1955.
10. Karabim, J. E.: Ruptured abdominal aortic aneurysm with retroperitoneal hemorrhage simulating acute appendicitis, Am. Surg. *18:* 1116 (Nov.) 1952.
11. Kirklin, J. W., and others: Surgical treatment of arteriosclerotic aneurysms of abdominal aorta, A.M.A. Arch. Surg. *67:* 632 (Nov.) 1953.
12. Farrar, T., and others: Surgical treatment of acute rupture of abdominal aortic aneurysms: report of 2 cases. Proc. Staff Mayo Clin. *31:* 299–304 (May) 1956.
13. Lowenberg, E. L.: Aneurysm of abdominal aorta: report of 2 cases treated by "cutis grafting", Angiology *1:* 396 (Oct.) 1950.
14. Milton, H.: Ligature of abdominal aorta for ruptured aneurysm of that vessel-death, Lancet *1:* 85, 1891.
15. Moore, in Finney, J. M. T.: Wiring of otherwise inoperable aneurysm, Ann. Surg. *55:* 661, 1912.
16. Schafer, P. W., and Hardin, C. A.: Use of temporary polythene shunt to permit occlusion, resection and frozen homologous graft replacement of vital vessel segments; laboratory and clinical study, Surgery *31:* 186 (Feb.) 1952.

SOME UNUSUAL OBSTRUCTIVE LESIONS OF THE URINARY TRACT

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Congenital or acquired obstructive lesions of the urinary tract comprise one of the most important groups of pathologic conditions in the field of urology. The urologist must be forever on the lookout for impediments to free urinary drainage at all levels of the urinary system, as these obstructions, if not symptomatic in themselves, certainly predispose to eventual infection and progressive destruction of functioning renal tissue. Early urologic investigation in these patients frequently affords the surgeon a good chance to prevent permanent renal damage and, by the same token, postponement of complete urinary tract investigation may result in the occurrence of irreversible damage, the burden of which must be shouldered by the physician.

As has been emphasized repeatedly in the recent literature, complete studies are possible in all age groups, regardless of sex, without undue trauma and with considerable safety. Cystoscopy, retrograde pyelography, cystography, and panendoscopy, even in the very young, can be accomplished with very little trauma and with considerable diagnostic accuracy in trained hands, and postponement of these indicated examinations until the infant or child is older is not justified. The tendency for single congenital anomalies of the genitourinary tract to be associated with other urinary defects makes it essential for the urologic examination to be complete in order to uncover all pathologic changes, as occasionally an unsuspected obstructive lesion may be the basic underlying cause for the patient's trouble. The following 4 cases of obstructive uropathy are presented in substantiation of the previous remarks.

CASE REPORTS

Case 1: M. B., a 54 year old white male executive, was completely asymptomatic and in apparent good health when a routine annual physical examination by his family doctor revealed a right upper quadrant mass. Ten years previously, urograms and cystoscopy had been done elsewhere following a single episode of hematuria, and he had been told his right kidney was enlarged. No treatment was given. Examination of the abdomen on Aug. 19, 1955, revealed a 15 cm., smooth, rounded, movable, nontender mass in the right abdomen and flank. Urinalysis was normal with no cellular elements in the urine, and blood count and serology were normal. Excretory urograms revealed a large but normally functioning left kidney. There was a large soft tissue mass over the right renal area obliterating the psoas margin, and there was no function through 20 minutes (fig. 1).

Cystoscopy showed a normal bladder. There was no efflux of urine on the right, and ureteral catheters met an impassable obstruction 2 cm. above the orifice. Retrograde injection of dye produced no filling on the right. It was thought that this was a large hydronephrosis, possibly due to ureterovesical stricture, but renal tumor or cyst could not be ruled out. Renal angiography was decided against, as we believed exploration should be carried out regardless of other findings.

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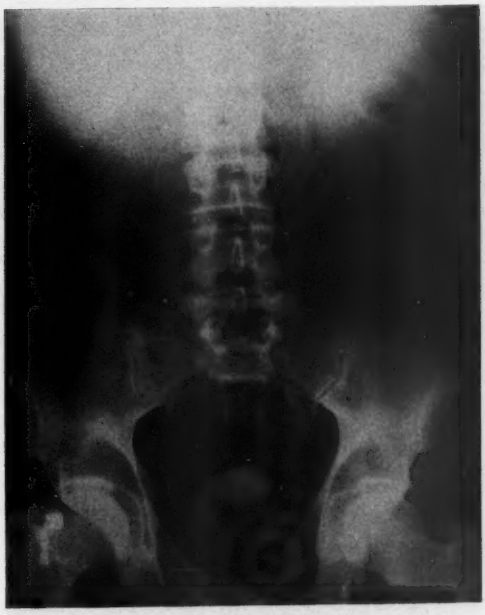

FIG. 1. Case 1, excretory urogram showing obliteration of psoas margin on right with no visible dye excretion.

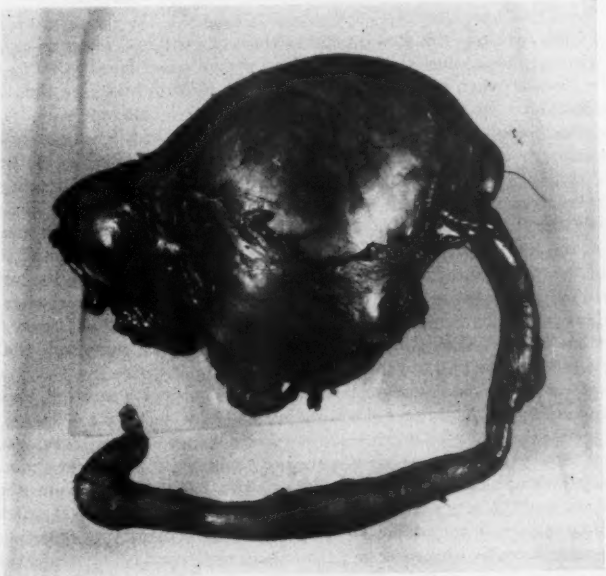

FIG. 2. Case 1, surgical specimen. Hydronephrosis and hydroureter. Note distal ureteral narrowing.

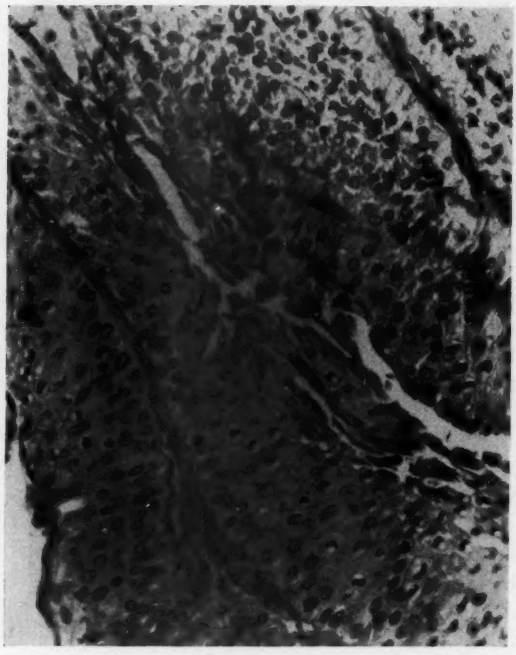

FIG. 3. Case 1, photomicrograph of ureteral tumor

On Aug. 26, 1955, the right kidney was exposed through the flank. The mass proved to be a tremendous hydronephrosis and hydroureter. The ureter was dilated down to a point 0.5 cm. above the bladder when there was found a dense filiform stricture (fig. 2). Nephroureterectomy was done, with excision of the ureter down to the bladder wall.

Pathologic study confirmed the surgical findings, but in addition there was found a primary ureteral tumor, 2 cm. in diameter, located within the dilated ureter and ending 1 cm. above the stricture. Microscopic sections showed a Grade I, noninfiltrating, papillary transitional cell carcinoma of the ureter (fig. 3). The ureter distal to the lesion was free of tumor, and there was no invasion deeper than the mucosa in any area. Since this lesion was unsuspected at the time of surgery, a bladder cuff was not removed. In view of the minimal malignancy of the tumor and the fact that the ureter was normal below the lesion, it was believed to be justifiable not to subject the patient to removal of the ureterovesical junction and a cuff of bladder at this time. The tendency for multicentric origin of tumors of urinary epithelium makes it essential to follow the status of the remaining kidney and the bladder at regular intervals, and this will be done.

Case 2: L. K., a 9 year old Japanese school boy, was born with multiple anomalies, including harelip, cleft palate, bilateral syndactylism and adactylism of the fingers. At age 2 a cleft palate repair was attempted but later broke down. At age 7 he first developed a urinary tract infection with intermittent fever, nausea and vomiting, dysuria and abdominal pain. Urine was loaded with pus and bacilli. He was found to have a very severe phimosis and was circumcised, but the attending surgeon suggested that urinary tract anomaly was a likely possibility, although the phimosis was causing some urinary obstruction in itself. He remained asymptomatic for a year and then again developed urinary infection that did not clear on antibiotics. Excretory urograms showed a solitary right kidney with mild dilatation of the pelvis and ureter, suggesting ureterovesical obstruction (fig. 4). Un-

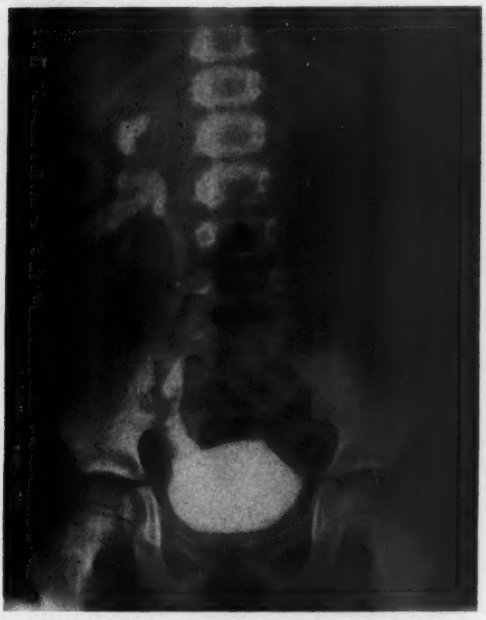

FIG. 4. Case 2, delayed excretory urogram showing compensatory hypertrophy of right kidney and evidence of distal ureteral obstruction.

usual dilatation of the distal ureter resembled the radiographic picture of ureterocele, but cystoscopy showed a normal right orifice. Retrograde pyelography on the right revealed a dilated and redundant ureter and probable ureterovesical obstruction, and the bladder appeared normal.

His infection was controlled by antibiotics, after which retrograde cystography, without anesthesia, showed a normal size bladder without reflux on the right and without trabeculation (fig. 5). On Aug. 24, 1955, exploration of the right ureter and bladder was done. There was a dense stricture of the distal right ureter just above the ureterovesical junction. The ureter was straightened, the redundant portion including the stricture excised, and the ureter reimplanted into the bladder. The anastomosis was splinted with a no. 8 straight catheter brought out from above alongside a suprapubic tube. The bladder neck was normal on inspection and was not altered. The splint and suprapubic tube were removed after 14 days and the bladder closed promptly on urethral catheter drainage. Although he is asymptomatic, the surgery is too recent to obtain significant follow-up radiographic studies.

Case 3: L. F., a 28 year old Japanese customs officer, was seen in the Surgery Department on Feb. 19, 1955, for prolapsed and painful hemorrhoids. These were treated surgically, but it was noted that his blood pressure was 158/66 and his urine showed 2 plus albumin. He was seen by the medical service several times after surgery and his blood pressure ranged between 150/90 and 140/84. Fundi, chest roentgenogram, electrocardiogram, and further urinalyses were all normal. No definitive therapy was given. In August 1955, he felt dizzy and his blood pressure was found to be 180/100, but then dropped to 150/90 and remained normal on subsequent examinations. Urinalysis again was normal. Excretory urogram was obtained as part of his routine studies for mild hypertension, and revealed a large but otherwise normal kidney on the left. There was no renal shadow and no dye excretion on the right (fig. 6).

Urologic consultation was requested, and on September 15, cystoscopy and panendoscopy

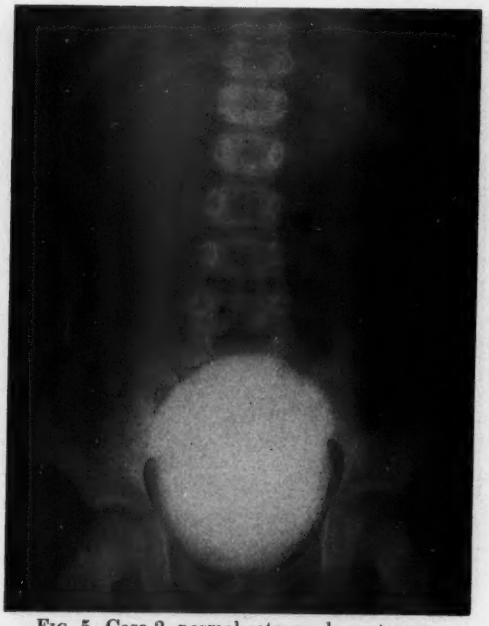

FIG. 5. Case 2, normal retrograde cystogram

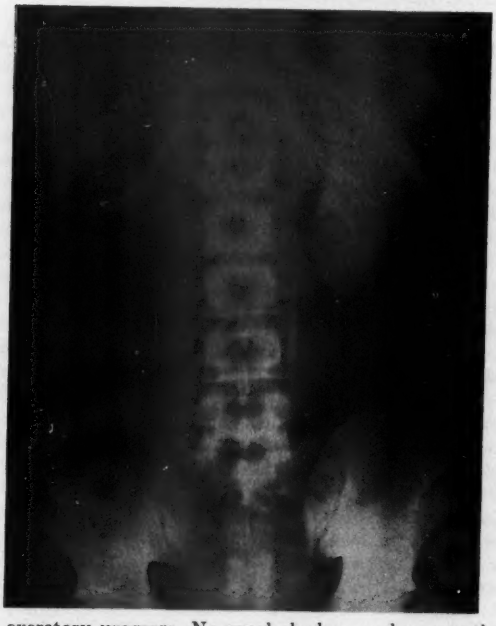

FIG. 6. Case 3, excretory urogram. No renal shadow or dye excretion on the right

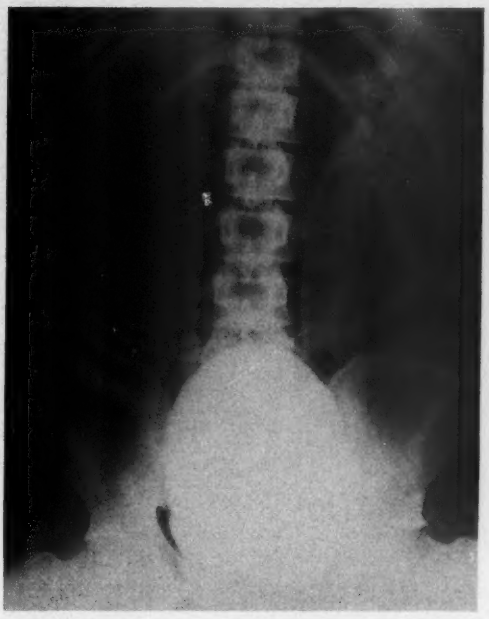

FIG. 7. Case 3, retrograde cystogram, anteroposterior view. Dilated bladder with ureteral reflux. Note small collection of dye lateral to L-5 on the right.

revealed a very large, trabeculated bladder. The left ureteral orifice was dilated. The right orifice was tremendously dilated and opened low on the trigone, almost at the vesical neck. A peculiar flap of tissue bulged into the internal vesical orifice and partly obliterated the right side of the prostatic urethra. Retrograde cystogram showed a huge bladder with a cpacity of 800 cc. (fig. 7). Reflux on the left outlined a normal ureter and pelvis. Oblique views also showed reflux on the right filling a tremendous, tortuous right ureter and a tiny hydronephrotic pelvic ectopic kidney lying over the right sacroiliac joint (fig. 8). Apparently the dilated intramural distal right ureter had produced bladder-neck obstruction. The prostate was small and not obstructing.

It was thought that his abnormal right kidney probably was not related to his mild hypertension, but that surgery was certainly indicated for urologic reasons alone. On Sept. 28, 1955, the bladder was explored through a suprapubic midline incision. The small ectopic kidney and dilated ureter were removed after extending the incision laterally and upward. Close inspection of the ureterovesical junction showed a large saccular dilatation of the intramural ureter which extended under the mucosa to undermine the vesical neck mucosa, simulating a large ureterocele. The lower flap of mucosa thus produced was partially obstructing the internal vesical orifice. This flap was removed and the edges sutured over (fig. 9). There was no primary bladder-neck contracture present, and the bladder-neck obstruction appeared to be completely relieved. The left ureter was not reimplanted as the upper tract was completely normal.

The patient made a good recovery, but it is still too soon after surgery at this time to do follow-up roentgenologic studies. His blood pressure remains at 140/90.

Case 4: M. S., a 6 year old Portuguese school boy, was seen by the general surgery service at the age of 5 years because of urinary burning and some intermittent trouble passing his urine. Physical examination was normal. Urine was not infected but showed moderate

FIG. 8. Case 3, retrograde cystogram, left oblique view. Reflux of dye outlining abnormal right ureter and pelvis.

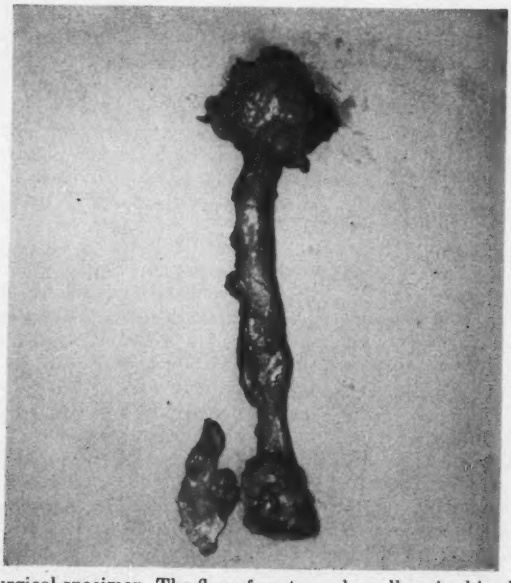

FIG. 9. Case 3, surgical specimen. The flap of ureterocele wall excised is shown on the left

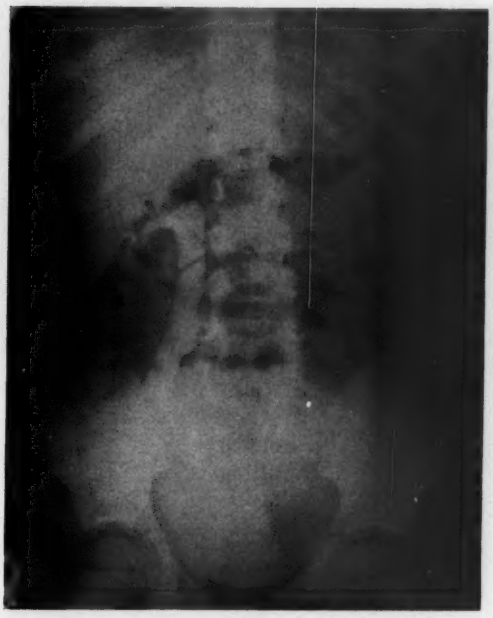

FIG. 10. Case 4, excretory urogram showing single right kidney with tortuous and dilated upper ureter.

microscopic hematuria. Excretory urograms were done and revealed no function on the left, and a definite renal shadow could not be seen on this side. The right kidney showed compensatory hypertrophy and there was moderate dilatation and redundancy of the upper ureter with slight dilatation of the renal pelvis (fig. 10). On April 28, 1954, cystoscopy was done. A left orifice could not be seen. There was no note on the cystoscopic appearance of the bladder. The right ureter was catheterized easily and retrograde pyeloureterograms revealed the same changes seen on excretory films (fig. 11). The distal right ureter was not particularly narrowed, but was not tortuous and redundant compared with the upper ureter.

The family elected to postpone surgery for the time being, and the boy was followed at intervals for one year. Occasional urinalyses showed microscopic hematuria in most instances. Two episodes of urinary infection were treated with antibiotics successfully. In general he appeared healthy and developed normally.

In August 1955, urologic consultation was obtained. Physical examination was normal. Urine showed no infection. Excretory urograms were repeated and again showed a solitary right kidney with a little increase in dilatation of the ureter and pelvis but still prompt function and good concentration. Nonprotein nitrogen was 23 mg. per cent. Although the impression up to this time had been possible ureterovesical obstruction, it was thought that bladder-neck obstruction had to be ruled out before surgery could be considered. Further study had to be postponed because of an upper respiratory infection associated with asthma, but this cleared satisfactorily on treatment.

On September 27, under pentothal-nitrous oxide anesthesia, panendoscopy revealed a very large trabeculated bladder with a diverticulum coming off each lateral wall. There was a very marked bladder-neck contracture with a high fibrous posterior vesical lip. Prostatic urethra was normal. Careful inspection failed to reveal the location of either ureteral

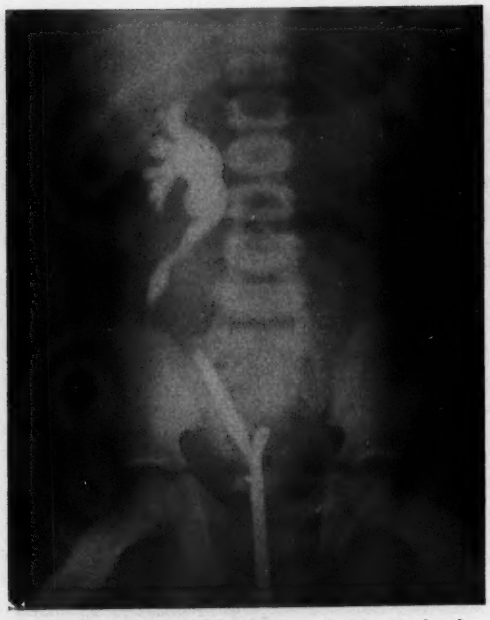

FIG. 11. Case 4, right retrograde pyelogram showing redundancy of ureter

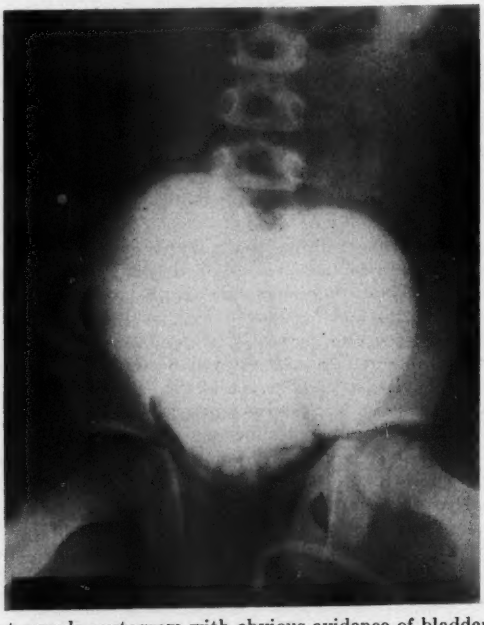

FIG. 12. Case 4, retrograde cystogram with obvious evidence of bladder neck obstruction

FIG. 13. Case 4, surgical specimen. Dilated left kidney and ureter

orifice. Retrograde cystogram demonstrated a huge bladder with a capacity of 500 cc. (fig. 12). There was a very large diverticulum on the left, almost as large as the bladder. No reflux was demonstrated. The diverticulum appeared to empty completely.

On Sept. 28, 1955, the bladder was explored through a suprapubic midline incision. As soon as the bladder was evacuated, a continuous jet of urine could be seen on the lower margin of the diverticulum on the left, marking the site of a left ureteral orifice not previously identified. The bladder neck showed a marked contracture which would not admit the tip of a finger. The right orifice could not be seen. The right ureter then was identified at the pelvic brim and opened with a tiny vertical incision. A no. 6 catheter could be passed down into the bladder and up into the kidney without meeting obstruction so the right side was left alone. The diverticulum was freed and removed, and during this dissection it became obvious that it had compressed and obstructed the distal left ureter. The left kidney and ureter were dissected free and removed through a lateral extension of the incision, and there was marked hydronephrotic atrophy of the kidney and moderate dilatation of the upper ureter (fig. 13). The bladder neck was handled with a deep posterior wedge and a Bradford Young type of closure of the anterior lip.

It was thought that all obstructive phenomena had been removed. Further exploration of the right ureter with straightening, shortening and reimplantation was considered but it was thought that sufficient surgery had been done by that time and that relief of the obstruction alone could result in return of the ureter to normal. This may be necessary in the future, but time will tell.

SUMMARY

Early urinary tract study in patients showing urinary infection, hematuria, or obstructive symptoms is almost always indicated, regardless of age.

Complete urologic evaluation may reveal an unsuspected lesion that is of greater importance than the known disease.

An unsuspected obstruction low in the urinary tract may be responsible for upper tract changes suggesting primary upper tract disease.

Four cases of urinary tract obstructive disease are presented illustrating these observations.

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BASAL CELL CARCINOMA OF THE ANUS: A CASE REPORT

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The majority of malignant anal lesions are epidermoid or squamous cell carcinomas while true basal cell carcinomas of the anus occur infrequently. Recently, however, Armitage and Smith[1], and Martineau, Saini and Clermont[2] have reported significant series of cases. The authors wish to report another case of basal cell carcinoma of the anus apparently successfully treated by roentgen therapy and to present some conclusions as to management of this malignancy.

CASE REPORT

A 73 year old white man, L. F. G., was admitted to the University of Virginia Hospital in November 1953 with a history of having had a growth around his anus of 2 to 3 years' duration. He had noticed minimal bleeding from this mass upon trauma during the past year and had considerable soreness, itching and discomfort about the anus.

Physical examination revealed an elderly, kyphotic white man in no acute distress. The lungs were emphysematous. The abdomen was negative except for a left inguinal hernia, which was easily reducible and did not descend into the scrotum. Palpable enlarged inguinal lymph nodes were noted bilaterally. Rectal examination demonstrated a cauliflower-like, granular, firm mass encircling the anus and arising from the mucocutaneous junction (fig. 1). Digital examination revealed that the mass apparently extended approximately 1 centimeter within the anus. It was the impression of several examiners that the mass involved the external sphincter. No other masses were felt.

The admission diagnosis was squamous cell carcinoma of the anus. Biopsy specimen was obtained after admission and was reported as basal cell carcinoma (fig. 2). Biopsy was repeated to confirm this diagnosis and again was reported as a basal cell carcinoma. Proctoscopic examination to the 6 inch level was negative except for the previously described anal lesion. A barium enema was reported as negative. The superficial inguinal lymph nodes were removed bilaterally under local anesthesia and were reported as showing marked hyperplastic lymphadenitis. Panendoscopic examination showed some enlargement of the lateral lobes of the prostate gland, and the bladder was essentially negative. Kahn test of the blood, urinalysis and blood counts were all within normal limits.

Following consultation with members of the Department of Radiology, and after a review of the literature on the subject, it was decided to attempt to treat this lesion with roentgen therapy alone, as even a local excision would necessitate sacrifice of the anal sphincter. Accordingly, he was started on deep roentgen therapy and received a total dosage of 3,600 roentgen units in air to the anal area. At first, his response to radiation therapy was slow, but when he was seen 6 weeks later, he had shown a remarkable regression of the tumor without disturbance in fecal control.

Examination 24 months after treatment revealed no evidence of local recurrence of the tumor (fig. 3). He appeared to be in excellent health and had no difficulty in controlling his bowel actions. Digital and proctoscopic examination to the 6 inch level, barium enema, roentgenograms of the bony pelvis, as well as panendoscopy were within normal limits as were routine blood counts and urinalysis. Roentgenograms of the chest revealed what was

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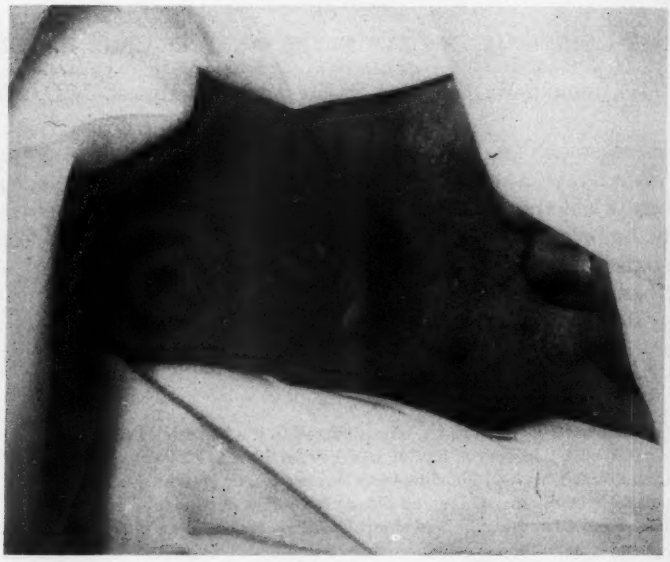

FIG. 1. L. F. G. Anal lesion, November 1953

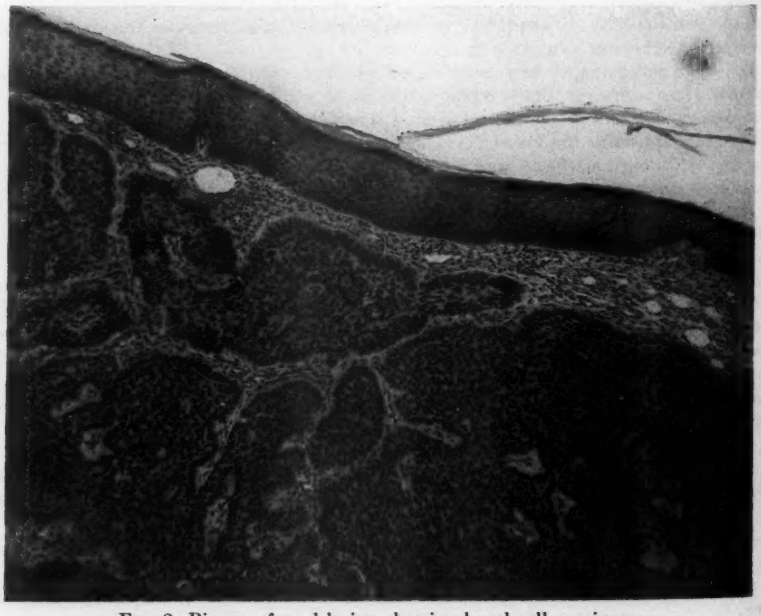

FIG. 2. Biopsy of anal lesion showing basal cell carcinoma

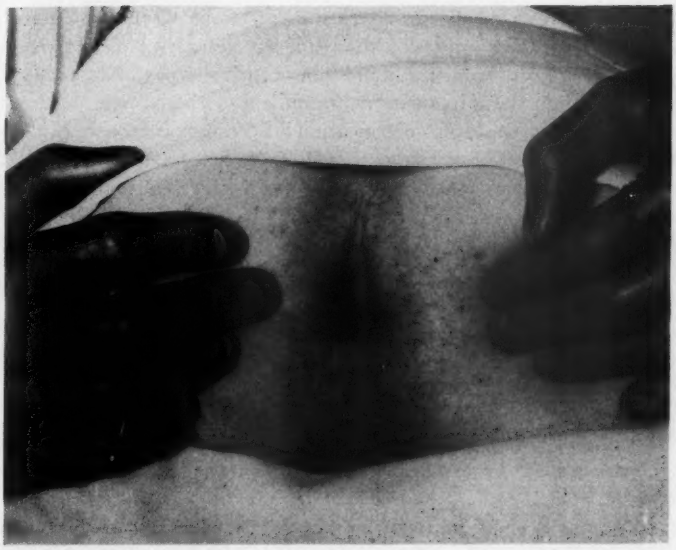

FIG. 3. Anal area, November 1955, showing radiation changes with no recurrence of tumor.

thought to be an osteolytic lesion in the posterior 7 centimeters of the right 8th rib. He was admitted to the hospital and under local anesthesia, the right 7th, 8th, 9th and 10th ribs were biopsied. Histologic examination of the decalcified material revealed no pathologic change. The changes noted on the roentgenograms probably represented demineralization of the right 8th rib. A transurethral resection was done and the report from the Department of Pathology was adenocarcinoma of the prostate, probably confined to the gland.

DISCUSSION

It is difficult to evaluate the methods used in treating this type of lesion. The confusion results from rarity of the lesion, from the difficulty in interpretation of the pathology of anal lesions and from the great variety of treatments that have been reported.

The importance of an accurate diagnosis cannot be over-emphasized. Epidermoid carcinomas may frequently show basal cell characteristics. Grinnell[2] has reported that 16 of his 49 cases of epidermoid carcinoma of the anus showed basal cell characteristics. Certainly, any so-called basal cell tumor of the anus must be examined carefully for this possibility. No doubt some of the reported cases showing metastases fall into this group, *i.e.*, they were not true basal cell carcinomas.

The variety of methods of treatment reported[1, 3] include local excision alone, excision with radiation, radiation alone, radiation with gland dissection, posterior resection of anus and rectum with and without node dissection, and Miles abdominoperineal resection.

The reported results of therapy are likewise difficult to evaluate. From the

reported cases, we can reach two conclusions: that basal cell carcinoma of the anus is a much less malignant lesion than epidermoid carcinoma of the anus; and second, that the results from the more conservative types of treatment are as good, or better, than those following more radical methods.

The authors believe that some clarification is necessary before we can approach this problem logically. Those lesions that have been treated radically with poor survivals are probably epidermoid carcinomas with basal cell characteristics. The lesions treated conservatively with good results are probably true basal cell carcinomas.

We would like to advocate the following principles in management of future cases.

1. Adequate and multiple biopsies to rule out the benign inflammatory lesions and the epidermoid carcinomas. If there are enlarged inguinal nodes, they should be removed for histologic examination and if a metastatic lesion is found, this is indication that the lesion is an epidermoid carcinoma.

2. If the lesion is a true basal cell carcinoma, it should be treated as such, that is with conservatism. True basal cell tumors rarely metastasize, but are locally invasive.

3. If the tumor is small and can be adequately excised with preservation of the sphincter, this should be the method of choice.

4. Should invasion of the sphincter have rendered excision impossible without sacrifice of the sphincter, external radiation should be attempted.

5. An abdominoperineal resection with a wide excision of anal skin should be done when a large lesion involves the entire anus and has destroyed the anal canal.

6. For local recurrences radiation should be employed, unless there has been invasion or destruction of the anal canal with loss of fecal continence, in which case proctectomy would become necessary.

CONCLUSIONS

A case of "true" basal cell carcinoma of the anus which was treated successfully with external radiation is reported.

The importance of an accurate histiologic diagnosis in all anal lesions has been emphasized.

Conservatism in treatment of these lesions is advocated unless the locally destructive effects of the tumor indicate a more radical approach.

A study of a large number of similar lesions is mandatory before a true evaluation of therapy can be made.

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REFERENCES

1. Armitage, G., and Smith, I.: Rodent ulcer of anus, Brit. J. Surg. *42:* 395 (Jan.) 1955.
2. Grinnell, R. S.: Analysis of 49 cases of squamous cell carcinoma of anus, Surg., Gynec. & Obst. *98:* 29 (Jan.) 1954.
3. Martineau, P. C., Saini, V. K., and Clermont, V.: Basal cell epithelioma of anus, J. Michigan State M. Soc. *53:* 390 (April) 1954.

VESICOCOLIC FISTULA COMPLICATING DIVERTICULITIS OF THE COLON

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Vesicocolic fistula is the most disabling complication of diverticulitis of the colon. This will increase with the frequent perforations, abscesses, and obstructions, which follow in their order of occurrence. Interest in this problem is centered in previously asymptomatic patients who incidentally are encountered with acute manifestations.

Roentgenographic evidence of diverticulosis of the colon occurs in from 3 to 4 per cent of the patients examined. Kocour[4] found that diverticula occurred in 1.8 per cent of 7,000 necropsies. It is well known that this disease occurs in patients past the age of 50 and usually in those who are moderately too markedly obese.

Diverticulitis occurs in approximately 10 per cent of all diverticulosis. The history of pain, frequently of an intermittent type, associated with varying degrees of constipation, a low grade fever and chills, is fairly suggestive of diverticulitis. Examination reveals abdominal tenderness in the left lower quadrant where generally approximately 75 per cent of diverticulitis is found. A confirmatory mass can be expected in long-standing disease, which further interferes with differentiation from malignancy. This is not easy and the diagnosis frequently can be made only by microscopic examination.

Intestinovesical fistulas are classified as traumatic, neoplastic and inflammatory in origin. Under normal circumstances traumatic forms are rare but during World War II many patients who were battle casualties with penetrating wounds were encountered with these lesions. Primary carcinoma involving the sigmoid or rectum accounts for the greatest number in the neoplastic group. Practically all of the inflammatory lesions are associated with diverticulitis, with or without abscess.

Ormond, Best and Klinger[7] stated that approximately 700 cases of vesicointestinal fistula had been reported and 25 additional ones were added. Mayo[6] reported 39 males and 7 females with the disease, which is the usual sex distribution. Lazarus and Marx[5] stated that inflammatory lesions occurred in 51 per cent of the patients who had fistulas, and diverticulitis was the primary cause in 65.8 per cent. Dick[2] recorded 1 case of intestinovesical fistula out of each 600 urologic patients admitted to the Lahey Clinic. In 1888, Crupps[8] made a complete report of this disease but only recently has the association of diverticulitis and its complications been recognized and appreciated.

In the patient with fistula there are varying degrees of discomfort associated

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Presented during The Richmond Assembly of The Southeastern Surgical Congress, March 12–15, 1956, Richmond, Virginia.

with chills and fever for long periods of time. Suprapubic pain and pneumaturia will be expected after the communication is complete. This is followed by fecaluria and a foul odor to the urine and in the late stages hematuria may be an accompaniment of these symptoms.

Many methods of establishment of the diagnosis are utilized. Mayo[6] stated that in 20 per cent of their cases the diagnosis could be confirmed by barium enema. The introduction of dyes in the gastrointestinal tract and the ingestion of charcoal may aid in demonstrating these lesions. Barium enema alone has been disappointing in proving conclusive evidence of such communications. This is understood in the early inflammatory period when the fistula may be extremely small and the introduction of barium would be difficult, although gas and liquid feces could readily pass into the bladder. On cystoscopic examination 84 per cent could be diagnosed, by the characteristic lesions located near the dome and on the posterior wall of the bladder. A cystogram sometimes can demonstrate the communication. Completion of the diagnosis may be made by the introduction of a ureteral catheter through this opening and suitable roentgenographic contrast media can be introduced. This may be somewhat difficult and perhaps hazardous.

There are two stages of the disease; those that develop incipiently and have indefinite symptoms and those that develop with cardinal manifestations at the onset. For months before the communication can be demonstrated the only feature may be subjective symptoms. This delay in recognition prolongs discomfort, increases morbidity, and may permit an overwhelming ascending urinary tract infection. It is believed that an early more aggressive approach should be undertaken to prevent this complication.

As was previously mentioned, considerable investigation is required, to exclude neoplasm, both on the gross and microscopic studies. Individuals past the age of 40 years are primarily effected, as is the case in those with a malignancy. Essentially the findings are those of varying degrees of inflammation, with or without abscess formation. There may be surprisingly few adhesions depending on the course and duration. Although not always present, involvement of the mesentery may occur in the advanced stages. The opening between the bladder and the colon usually are not large and often cannot be demonstrated on cystoscopic examination. Spontaneous closure of the opening can occur but is ex-extremely rare.

Treatment in recent years has been well established after confirmation of the diagnosis of a benign inflammatory fistula. Essentially the methods of treatment are: (1) a single stage resection, with or without establishment of a proximal colostomy and (2) a preliminary cecostomy or colostomy for decompression with ultimate resection of the involved area of the bladder and colon followed by restoration of the continuity of the organs. A large number can be treated in one stage with the use of proper preoperative preparation and adequate resection. Many patients who have marked inflammatory changes should have either preliminary transverse colostomy, preferably followed by resection of the involved area, or a supplemental colostomy at the time of the primary resection.

The former procedure is much safer and lessens the likelihood of unpleasant morbidity and would greatly minimize the mortality.

Judd[3] has emphasized early resection in the treatment of recurrent sigmoid diverticulitis. When travel in remote areas is contemplated removal of the diseased colon is advised during a quiescent period. This is recommended particularly when diverticulitis is associated with bladder symptoms. Sixty-eight patients were so treated by a single stage operation. The indications were either a mass, hemorrhage, obstruction, or fistula formation. More and more stress is placed on the importance of prophylactic resection as a measure to avoid the development of this extreme disability.

Barnes and Hill[1] reported 4 recurrences following a single stage repair of intestinovesical fistula. There were no recurrences in 6 patients who had a preliminary colostomy. Although disability may be prolonged, it is believed that stage operations are more satisfactory and that an associated colostomy will decrease the risk.

CASE REPORTS

Case 1. H. J. C. A 75 year old white man was admitted to Emory University Hospital on Nov. 7, 1954, with a history of passage of gas and feces through his penis. He had marked weakness, and headache of several weeks duration. A barium enema demonstrated many diverticula in the sigmoid colon but a communication in the bladder could not be seen.

He had marked angina pectoris and poor exertion tolerance. Repeated electrocardiograms failed to demonstrate evidence of myocardial infarction.

At cystoscopy a necrotic area was seen on the posterior bladder wall. A biopsy section was obtained and the microscopic examination was thought to show a squamous cell carcinoma of the bladder.

A laparotomy was performed on Nov. 24, 1954, which revealed a fistulous communication between a ruptured sigmoid diverticulum and the dome of the bladder. The involved area of the bladder and sigmoid colon was widely resected. Although chronic inflammatory disease was present microscopic examination of tissue did not substantiate the previous diagnosis of malignancy. His postoperative convalescence was normal.

He was readmitted to the Hospital on May 3, 1955, semicomatose, with a history of 3 days prior to admission having developed fever, vomiting, diarrhea, and lower abdominal pain. His blood pressure was 210/110. His abdomen became progressively distended and he died 48 hours after admission to the Hospital.

Postmortem examination revealed thrombosis of the superior mesenteric artery with necrosis of the small intestines. A mural thrombus was found on the wall of the aorta and multiple thromboses of the left internal iliac artery. There was an infarction of the left kidney. The urinary bladder and large intestine were normal. No evidence of malignancy could be demonstrated in the urinary bladder or the sigmoid colon.

Case 2. C. A. D. A 58 year old white man was admitted to the Atlanta Veterans Hospital on April 18, 1955. Onset of present illness began 1 month before when he developed dysuria, and passage of gas through his penis after urination.

In the fall of 1954, the patient had episodes of frequent diarrhea and low sacral discomfort. On admission to the Hospital physical examination was essentially negative, laboratory findings were normal, and roentgenogram of his chest revealed pulmonary fibrosis. Barium enema demonstrated extensive diverticulosis in the sigmoid colon, and a fistula into his urinary bladder. Retrograde pyelogram revealed no abnormalities of the ureters or kidneys. Cystogram did not demonstrate the communication between the bladder and the colon. On cystoscopic examination a lesion was found on the left posterior wall of the blad-

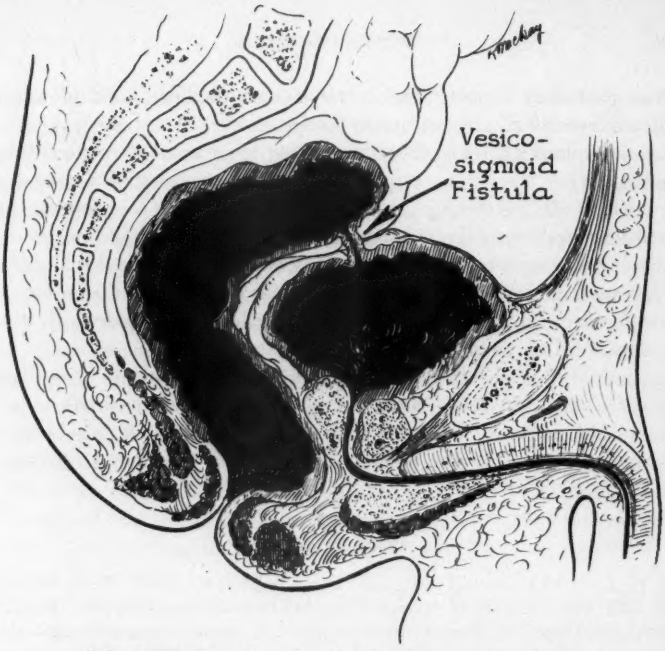

FIG. 1. Diagram of vesicocolic fistula as seen in Case 1

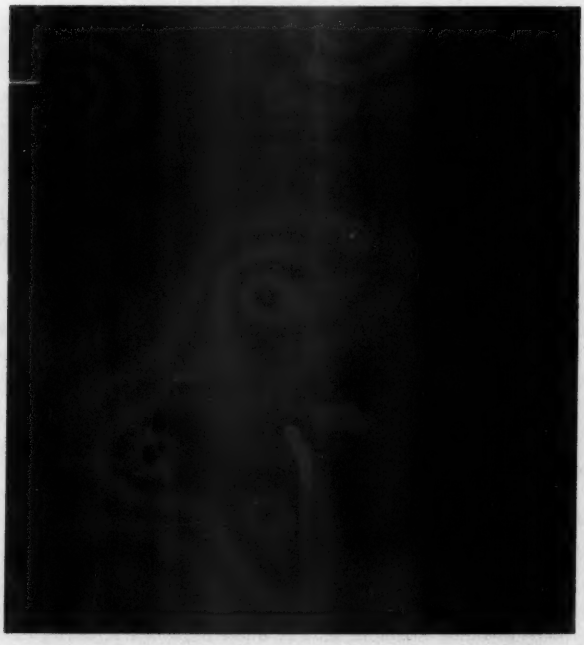

FIG. 2. Roentgenogram demonstrating ureteral catheter passed through vesicocolic fistula at cystoscopy as seen in Case 3.

FIG. 3. Roentgenogram demonstrating marked degree of diverticulitis with extensive scarring, which represents excellent opportunity for production of vesicocolic fistula.

der. Biopsies sections were obtained which were thought to show grade 1 transitional cell carcinoma. Immediate preparation was made for resection of the colon and bladder wall. On exploration of the abdomen on May 3, 1955, a communication was found between the midsigmoid colon and the upper left dome of the bladder. There was minimal inflammatory reaction and no gross evidence of malignancy.

Resection was done by excising a wide area of the bladder wall as well as a 17 cm. section of the sigmoid colon. No demonstrable nodes were found. Microscopic examination of the bladder wall showed acute and chronic inflammation with no evidence of malignancy. The bladder was closed and a suprapubic tube was left in the bladder. An end to end anastomosis of the sigmoid colon was done. Convalescence following this procedure was uninterrupted and at the present he has no symptoms.

Case 3. R. M. This patient, aged 50, was seen and treated by Doctors Thomas Florence and Lamar Glass, in October 1955, at which time he gave a history of pain in his lower abdomen of about 10 years duration. In 1949, he had severe pain in his lower abdomen and

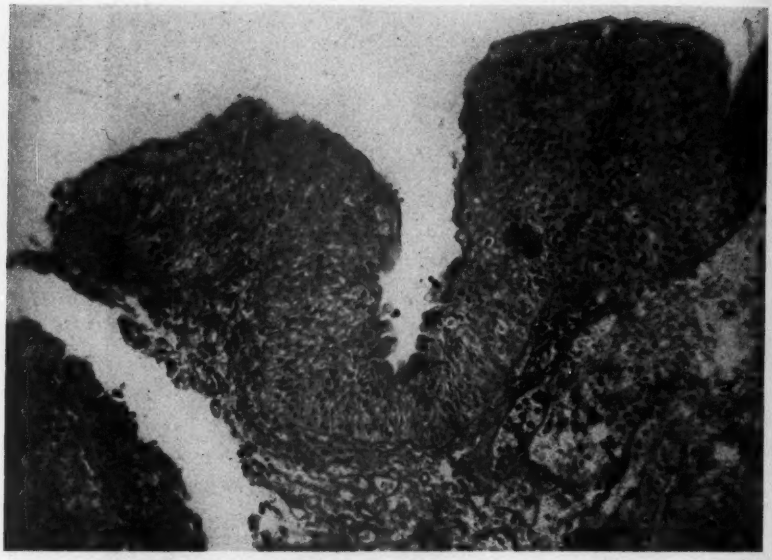

FIG. 4. Photomicrograph of biopsy from bladder of Case 1, demonstrating extreme squamous metaplasia and chronic inflammation.

diagnosis of diverticular abscess was made. This was treated conservatively and his symptoms cleared up. However, periodically he continued to have vague discomfort in his lower abdomen.

Two weeks prior to admission to Crawford Long Hospital there was increased frequency of urination. This was followed by passage of a large quantity of cloudy urine and gas through the urethra. At cystoscopy, induration was seen in the dome of his bladder. A definite opening could not be seen in this inflammatory mass but a no. 5 ureteral catheter was gently introduced into this mass. An immediate film revealed that it was within the lumen of the sigmoid colon. Contrast media then was introduced into the colon through the catheter.

Preparation was made for exploratory laparotomy and on Oct. 10, 1955, an inflammatory mass was found to be arising from the sigmoid diverticulitis. A small area of communication was demonstrated. This entire area was resected and an immediate end to end anastomosis was done. The postoperative course was without event and the patient has had no symptoms since.

DISCUSSION

Three cases are reported with vesicocolic fistula that developed following acute perforated diverticulitis. This development may be associated with a stormy inflammatory process within the abdomen or may develop with a mild lower abdominal discomfort. These also may be associated with varying degrees of obstruction or with hemorrhage. A large number have all the symptoms referable to the urinary tract. The outstanding features are those of pneumaturia, pyuria, and in the later stages hematuria. Ascending infection can occur but usually therapy has been instituted before this develops.

At cystoscopy 2 of these cases were diagnosed as epidermoid carcinoma involving the wall of the bladder. This opinion was based on the gross appearance and the microscopic examination of the tissue removed. The histologic features, in addition to marked metaplasia of the squamous epithelium, were highly suggestive of low grade carcinoma of the bladder. In the presence of an associated diverticulitis and with the suspicion of malignancy the treatment would be unaltered in the two conditions. In obtaining a biopsy specimen during cystoscopy an adequate section should be removed for satisfactory evaluation.

SUMMARY

Vesicocolic fistula complicating diverticulitis occurs sufficiently often to warrant suspicion in individuals with urologic symptoms.

This condition most frequently occurs in obese men past the age of 40 years.

It may develop with no previous evidence of diverticulitis or it may be associated with varying degrees of longstanding inflammatory changes. Diagnosis can be made by barium enema, cystoscopy, a cystogram and frequently with the use of oral dyes.

The pathologic appearance may be like that seen in low grade epidermoid carcinoma involving the bladder. For that reason, when at cystoscopic examination such evidence is found a barium enema should be done to exclude diverticula of the colon.

Resection of the involved segment of the bladder and the colon constitute the necessary means of correction of this disease. Preliminary colostomy is best instituted in those associated with marked acute inflammation. Primary resection may be safely performed in the chronic stage.

Prophylactic resection of the colon with acute or chronic diverticulitis, with obstruction or hemorrhage, should prevent this type fistula. This has been made possible by the use of adequate preoperative antibiotics, sufficient fluids, electrolytes, and blood.

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REFERENCES

1. Barnes, R. W., and Hill, M. R.: Intestinovesical fistula, J.A.M.A. *133:* 456 (Feb. 15) 1947.
2. Dick, V. S.: Intestinovesical fistula, Lahey Clinic Bull. *6:* 21 (July) 1948.
3. Judd, E. S., Jr.: Prophylactic resection for recurrent sigmoidal diverticulitis, Surg., Gynec. & Obst. *101:* 105 (July) 1955.
4. Kocour, E. J.: Diverticulosis of colon; its incidence in 700 consecutive autopsies with reference to its complications, Am. J. Surg. *37:* 443 (Sept.) 1937.
5. Lazarus, J. A., and Marks, M. S.: Vesicointestinal fistula-actual and incipient; early diagnosis and treatment, Am. J. Surg. *59:* 526 (March) 1943.
6. Mayo, C. W., and Blunt, C. P.: Vesicosigmoidal fistulas complicating diverticulitis, Surg., Gynec. & Obst. *91:* 612 (Nov.) 1950.
7. Ormond, J. K., Best, J. W., and Klinger, M. E.: Vesicointestinal fistulas, Surg., Gynec. & Obst. *89:* 411 (Oct.) 1949.
8. Senger, F. L.: Vesicocolic fistula complicating diverticulitis; case report, J. Urol. *61:* 437 (Feb.) 1949.

OMENTAL DERMOID CYST

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Dermoid cysts of the omentum and mesentery are very rare tumors. From the literature, which is scarce and confusing, it is impossible to determine the incidence of omental dermoids. Most authors have reported a single case and a few have attempted to total the previously reported ones. In 1928 Mumey[10] collected 15 cases (2 in males) and stated that Meckel in 1815 described the first omental dermoid cyst. In 1931 Lazarus and Rosenthal[5] reported 16 dermoids of the omentum and claimed that Lebert in 1734 was the first to report such a case. Judd and Fulcher[4] in 1933 collected 17 cases of omental dermoids. In a search of records of the Mayo Clinic from 1911 to 1949 Beahrs and Dockerty[1] found 29 cases of omental cysts, but none were dermoids.

Exclusive of the fields of gynecology and urology, dermoid cysts of the abdomen have been reported in the omentum, mesentery, stomach, pancreas, and retroperitoneum. Prior to December of 1931 Judd and Fulcher found only 7 abdominal dermoids exclusive of the omentum and gynecologic and urologic fields. In 1935 Meyer and Shapiro[8] collected from the literature 34 cases of abdominal dermoids, exclusive of gynecologic and urologic material. The following year in 1936 MacDermott[7] found 5 additional cases for a total of 39. Montgomery and Morest[9] in 1934 claimed to report the ninth proved case of mesenteric dermoid cyst and stated that dermoid cyst was first described in 1852 by Lebert. In 1938 Penberthy and Brownson[11] stated that only 15 cases of mesenteric cysts had been reported in the literature and in 1944 James[3] collected a total of 16 dermoid cysts of the mesentery.

From the reports in the literature it appears reasonable to assume that less than 25 cases of omental dermoids have been reported. The same estimate also might be made for mesenteric dermoids. Exclusive of gynecologic and urologic material, there probably have been less than 50 reports of abdominal dermoid cysts.

A dermoid cyst is lined by epithelium which may be destroyed by intracystic pressure and consists of skin, dermal glands, hair, sebaceous material, and sometimes of other ectodermal structures as teeth and bone. The finding of such a tumor in the omentum can be explained on the basis of misplaced embryonal ectoderm. Ewing[2] stated that dermoids commonly originate by inclusion of a portion of ectoderm during closure of embryonal fissures, at a point of union of ectoderm with other structures, along the course of ectodermal invaginations, or from persistent embryonal ectodermal structures. Lexer and Bevan[6] believed omental dermoids to be due to ectodermal invagination during closure of the abdominal midline fissure.

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Embryologically the omentum and mesentery develop in the dorsal portion of the celom from the mesogastrium. The development of these structures is in close proximity to the Wolffian and Mullerian ducts. It is believed that ectodermal cells of these ducts become attached to the mesogastrium to later give rise to dermoids of the omentum or mesentery.

Another etiologic theory of such tumors is that they are implantation cysts of the ovary. Epithelial cells may become attached to the omentum or mesentery as a result of traumatic rupture of an ovarian dermoid, or a dermoid may become detached from the ovary by the twisting or stretching of its ovarian attachment and the development of a blood supply through adhesions to the omentum or mesentery. Others have postulated that a supernumerary ovary might give rise to such tumors. It also has been suggested that ovulation, menstruation, or pregnancy might in some way be related to the origin of these tumors, because their incidence is much greater in women. Ewing stated that traumatic origin of dermoids has been demonstrated and that many authors have experimentally produced dermoid cysts by implanting skin.

Dermoids of the omentum and mesentery occur mostly in women although a few have been found in men. They have been reported in all ages and in both white and Negroes. They may be single or multiple and unilocular or multilocular. Dermoid cysts have been found in various portions of the omentum and mesentery including the gastro-splenic and gastro-hepatic ligaments and the lesser omental bursa. Some reports have stated that the ovaries were normal and in others an ovarian dermoid also was present. In the latter instance the omental or mesenteric dermoid may be free of the ovarian dermoid or attached to it. Omental and mesenteric dermoid cysts may grow to various sizes.

Preoperative diagnoses of omental or mesenteric dermoids have been rare. A radiologic finding of calcareous deposits, teeth, or bone might lead to an accurate diagnosis prior to surgery. A great variety of other diagnoses have been made, in one instance carcinoma of the stomach. Some of these cysts have been found accidentally during surgery for other reasons.

The symptoms are not pathognomonic but may be very vague and bizarre and usually are due to complications. If a tumor is discernible it may present various findings. The complications are mechanical pressure on surrounding organs or vessels, adhesions to neighboring structures, infection or suppuration, torsion and intestinal obstruction. Hemorrhage is unusual. Rupture of the cyst is rare and papillary ingrowths and neoplastic changes have not been reported.

The treatment is surgical excision with bowel resection if necessary. Marsupialization should be used only when enucleation technically is impossible. Recurrence following extirpation has not be reported.

CASE REPORT

A 69 year old white widow was admitted to Garfield Memorial Hospital on March 20, 1955, at 2 a. m. She was known to have had diabetes for 4 years, but had been well controlled with a daily dose of 16 units of N. P. H. Insulin. One and a half years previously she had a cervical laminectomy for radiculitis due to hypertrophic spur formation. She had hypertension and a year before had recovered from a partial paralysis of the left arm and

leg. Three months previously she had been hospitalized for thrombophlebitis of the left leg and hypertension of 220/110.

Following an automobile trip on March 18, 1955 she suddenly developed pain beneath the right breast, tightness in her chest, dyspnea, nausea, and vomiting. The following day the pain persisted at the right-costal margin and radiated to her back and right shoulder. The nausea and vomiting continued. She complained of chilly sensations and slight sweating, but had no fever.

Examination disclosed a stout woman, weighing 159 pounds, with good color. Her temperature was 98 F., pulse 100, respirations 24, and blood pressure 160/90. There were a few moist rales in both lung bases. The heart sounds were regular but distant. The abdomen was soft but tender, especially over the gallbladder, and the gallbladder was thought to be palpable. The blood sugar was 280 mg. per cent, hemoglobin 13 Gm., and the hematocrit 41 per cent. The white blood cell count was 21,000 per cu. mm. with segmented forms 81 per cent, band forms 3 per cent, and lymphocytes 16 per cent.

On the evening of admission the right abdominal pain and vomiting continued and the abdominal findings were unchanged. Her temperature was 99.4 F., pulse 114, and respirations 20. The white blood cell count had risen to 33,300 per cu. mm. with segmented forms 88 per cent, band forms 7 per cent, and lymphocytes 5 per cent.

On the following day the patient continued to be acutely ill with no significant change in symptoms or physical findings. Her temperature was 99 F., pulse 114, respirations 24, and blood pressure 150/108. The blood sugar was 245 mg. per cent; serum amylase 38 mg. per cent; urea nitrogen 22 mg. per cent; and serum sodium 138.5 mEq/L. An electrocardiogram showed sinus tachycardia and left ventricular hypertrophy. A chest roentgenogram disclosed some pulmonary congestion, the heart at the upper limits of normal, and an elongated and dilated aorta. A roentgenologic survey of the abdomen showed a large calcific density in the right-upper quadrant above the iliac crest having the appearance of a calcified gallbladder and calcification of the splenic artery.

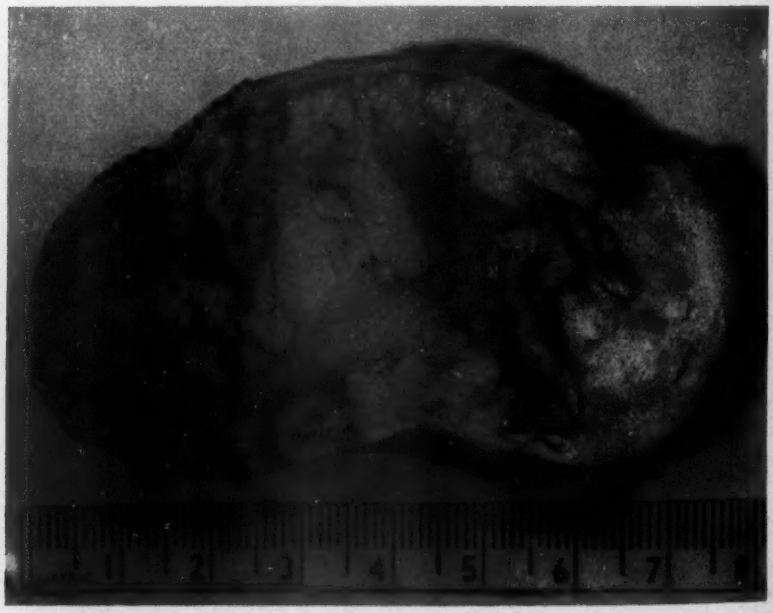

FIG. 1. Photograph of gross pathologic specimen of omental dermoid

On the afternoon of March 21 a laparotomy was performed through a right-subcostal incision. The gallbladder was normal. An oval-shaped tumor measuring 8 by 5 by 4 cm. was found on the right side in the omentum and attached to no other structure. This tumor was removed. Exploration failed to reveal additional pathology and the abdomen was closed without drainage. The patient's condition was considered grave during the evening following surgery. The temperature was 101 F., pulse 120, respirations 24, and blood pressure 186/120. Her heart sounds had improved in quality and her urinary output was considered satisfactory. At 1:30 a. m. on March 22 she developed cyanosis and death occurred.

The omental tumor was 8 by 5 by 4 cm., hard in some areas and firm in others, and the surface was irregular, mottled and reddish pink. The cyst wall averaged 0.2 cm. in thickness and was partially calcified. The lumen contained yellowish-gray, greasy material interspersed with a large amount of hair. The pathologic diagnosis was dermoid with calcification in its wall.

At autopsy the pericardium was distended and contained 500 cc. of fluid and clotted blood. The heart weighed 390 Gm. On the anterior wall of the left ventricle close to the interventricular septum and 2.5 cm. above the apex was a slit-like perforation measuring 1 cm. Both coronary arteries showed atheromatous plaques but their lumina were patent and no thrombi were found. Other pathologic findings were recent and remote myocardial infarction, generalized arteriosclerosis, bilateral pleural effusion, a 1 cm. colloid adenoma of the right thyroid lobe, and chronic passive congestion of the lungs, liver, and spleen.

DISCUSSION

The primary reason for this report is to record a case of omental dermoid cyst which was not connected to the ovaries and in which the ovaries presented no disease nor neoplasm.

Of secondary importance this report illustrates the difficulty in differential diagnosis between acute cholecystitis and coronary heart disease with which the abdominal surgeon is occasionally confronted. This patient's acute illness began with coronary heart disease, possibly severe spasm. At autopsy the coronary arteries did not contain thrombi, but did show atheromatous plaques and the myocardium showed recent and remote infarction. Degeneration of the left ventricular wall due to recent infarction lead to perforation with resultant cardiac tamponade and death. The omental dermoid cyst was an incidental finding at surgery and was not related to the acute illness, nor to the patient's demise.

The failure of the electrocardiogram to demonstrate evidence of coronary disease or myocardial infarction; the palpable mass which was mistaken for the gallbladder, but was in fact the dermoid; and the roentgenologic finding of a partially calcified mass in the upper-right quadrant which was also mistaken for the gallbladder, but again due to the dermoid, mis-led all the physicians who saw the patient to the erroneous diagnosis of acute cholecystitis for which the operation was performed.

SUMMARY

The incidence, etiology, diagnosis, and treatment of dermoid cysts of the omentum and mesentery have been presented and a case of omental dermoid has been reported.

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REFERENCES

1. Beahrs, O. H., and Dockerty, M. B.: Primary omental cysts of clinical importance, Surg., Clin. North America *30:* 1073 (Aug.) 1950.
2. Ewing, J.: Neoplastic Diseases, 4th. ed., Philadelphia, W. B. Saunders Co., 1940 p. 1063.
3. James, C. F.: Dermoid cysts of mesentery-case report, Am. J. Surg. *65:* 116 (July) 1944.
4. Judd, E. S., and Fulcher, O. H.: Dermoid cysts of abdomen, Surg. Clin. North America *13:* 835 (Aug.) 1933.
5. Lazarus, J. A., and Rosenthal, A. A.: Synchronous dermoid cyst of great omentum and ovary, Ann. Surg. *93:* 1269 (June) 1931.
6. Lexer, E., and Bevan, A. D.: General Surgery, New York, D. Appleton Century Co., 1908, p. 915.
7. MacDermott, E. N.: Suppurating dermoid cyst of gastrohepatic omentum, Lancet *1:* 898 (April 18) 1936.
8. Meyer, K., and Shapiro, P.: Dermoid cyst of lesser omental bursa, Am. J. Surg. *27:* 551 (March) 1935.
9. Montgomery, J. G., and Morest, F. S.: Dermoid cysts of mesentery, J. Missouri M. A. *31:* 456 (Dec.) 1934.
10. Mumey, N.: Dermoid cyst of great omentum, Am. J. Surg. *5:* 56 (July) 1928.
11. Penberthy, G. C., and Brownson, K. M.: Dermoid cysts of mesentery, Ann. Surg. *107:* 566 (April) 1938.

ACCURACY IN THE DIAGNOSIS OF JAUNDICE WITH PARTICULAR REFERENCE TO THE INTRAVENOUS GALACTOSE TOLERANCE TEST*

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Unnecessary surgical exploration of the jaundiced patient is accompanied by an increased morbidity and mortality rate. The literature contains a wealth of reports on persistent attempts to improve our diagnostic accuracy. There still, however, remains a significant deficit. The purpose of this report is to describe the diagnostic accuracy and experience with 50 jaundiced patients with histologic proof of etiology, and to encourage the more general use of the intravenous galactose tolerance test.

Several authors[1, 9, 11, 18], have reported their accuracy in the differential diagnosis of jaundice. Ivy[9] pointed out that this accuracy is about 60 per cent at the bedside. Watson[20] stated that laboratory tests can raise this accuracy to from 85 to 90 per cent. Maclagan[11] on the basis of the serum alkaline phosphatase and the thymol turbidity tests, stated that his diagnostic accuracy was from 65 to 79 per cent.

The intravenous galactose tolerance test has not been generally used. This is partly due to the fact that the oral test proved to be so disappointing. The intravenous test, however, in our experience has been accurate and helpful in the differential diagnosis of jaundice. It is of particular value in the hepatocellular type of jaundice and in such cases its value compares favorably with the value of the bromsulphalein excretion test in nonjaundiced liver disease. In 1941 Basset, Althausen and Coltrin[1] called attention to the intravenous galactose tolerance test as of value in differentiating obstructive from parenchymatous jaundice. Sherlock[18] in applying the test to patients with progressive obstructive jaundice, found that the test became positive only in the terminal stages of the disease. There also is the clinical experience of Ivy[9], Zieve[22] and Colcher and his associates[4] which indicated that there is relatively little impairment in hepatic metabolism of galactose in patients with obstructive jaundice and frequently considerable impairment in patients with hepatocellular disease.

CASE MATERIAL AND METHODS

The series to be described in this report includes 50 jaundiced patients from the Denver General Hospital, the Colorado General Hospital and the Denver Veterans Administration Hospital. The cases are unselected in the sense that they were seen consecutively by one of the authors (C. A. M.) during the past 18 months and constitute all of the jaundiced patients seen during that period who had histologic proof of the etiology of their jaundice. The series is, however, weighted with a high per cent of diagnostic problems which required unusually

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thorough work-up. The galactose*, alkaline phosphatase[3] and the thymol turbidity tests[6] were all done by one well trained research chemist with a known per cent error for each determination. The bilirubin and nonprotein nitrogen were performed in each hospital chemical laboratory by the Malloy-Evelyn[14] and micro-Kjeldahl[16] methods of analysis respectively. The normal value of the serum bilirubin was 1.0 mg. per cent or less. The normal value of the serum non protein nitrogen was 40 mg. per cent or less.

To determine the normal values for the blood galactose, serum alkaline phosphatase and serum thymol turbidity tests, blood samples were obtained from 100 hospital patients without liver disease. Eighty-seven of these patients showed a 75 minute galactose residual of less than 15 mg. per cent and no evidence of renal impairment. Thirteen patients showed 75 minute residuals of between 15 and 30 mg. per cent. All 13 had evidence of significantly impaired renal function. Actually, however, impaired renal function and azotemia does not necessarily nullify the galactose result since a serum nonprotein nitrogen from the upper limit of normal (40 mg. per cent) to 60 mg. per cent was associated with a 75 minute galactose residual of less than 30 mg. per cent. Thus a slightly elevated serum nonprotein nitrogen (45 to 55 mg. per cent) and a high abnormal galactose residual (50 to 90 mg. per cent) is indicative of hepatocellular damage and may be interpreted as such in our experience.

Ninety-nine of the patients showed a serum level of alkaline phosphatase of 5.0 Bodansky units or less. The one abnormal high was in a patient with recent acute pancreatitis. Ninety-seven of the patients showed serum thymol turbidity of 5.0 Maclagan ($CuSO_4$) units or less. The 3 abnormal results were in 2 patients with peptic ulcer and 1 patient with an appendiceal abscess.

RESULTS

Interpretation of the intravenous galactose tolerance test depends upon the fact that a given amount of galactose is converted to glycogen by the liver in a certain period of time with normal renal function. Thus an abnormally high 75 minute residual of blood galactose in the presence of normal renal function is an indication of impairment of hepatocellular function.

Interpretation of the serum alkaline phosphatase test depends upon the following physio-pathology. In the presence of biliary obstruction, the alkaline phosphatase which is made by the liver cells is excreted into the bile which is returned to the blood stream via lymphatics and gives an elevated serum alkaline phosphatase determination[7, 19]. Thus an abnormally high serum alkaline phosphatase (if unusual sources may be ruled out such as Paget's disease) indicates obstruction

* The intravenous galactose tolerance test[10] was performed as follows: After obtaining a control sample of blood in a fasting and carefully weighed patient, 1 cc. of a 50 per cent solution of galactose per kilogram body weight was injected intravenously. A second sample was drawn 75 minutes later. Chemical analyses were done according to the methods of Raymond and Blanco[17] and Benedict[2]. The normal value for the 75 minute blood sample in our laboratory is 14 mg. per 100 cc. or less as determined in 100 patients with normal liver function. A serum nonprotein nitrogen should be obtained on the same day. Since galactose is partly excreted through the kidneys[4, 5, 8] it is important to know whether significant impairment of renal function exists. We have used the serum nonprotein nitrogen test as an indicator of such.

somewhere in the biliary system. The interpretation of the thymol turbidity test depends upon the empirical observation of Maclagan that a solution of thymol iodide added to serum results in turbidity. Abnormal turbidity depends upon an elevated gamma globulin[12]. Since Watson and Rappaport[21], Meyer and Popper[15] and Maclagan[13] have shown that an elevation in gamma globulin is associated with hepatocellular damage the thymol turbidity test thus indicates impaired hepatocellular function.

Before discussing results it is important to emphasize that liver function tests indicate impairment of a specific function and do not give the clinician a pathologic diagnosis. The degree of impairment should be interpreted in the light of known pathology of specific disease and thus aid in the diagnosis. This point may seem trite to experienced clinicians but in our experience such emphasis has facilitated a better understanding of the underlying hepatic pathology and thus led to a more accurate diagnosis.

The relevant data from the 50 patients in this series are tabulated in tables I and II. Table I includes the 24 patients with hepatocellular jaundice. Table II includes the 26 patients with obstructive jaundice. Each of these two large groups are subdivided into a group (Group A) in which interpretation of the laboratory tests is indicative of either hepatocellular jaundice or obstructive jaundice, and a group (Group B) in which the interpretation is not indicative. This arrangement is made to emphasize the diagnostic value of these laboratory tests. The tests are indicative of the correct final diagnosis in 84 per cent of the cases.

Comparison of the diagnosis from the history and physical examination with the final diagnosis shows that in the total series of 50 patients the diagnosis by history and physical examination was 70 per cent accurate. This accuracy was little better than the toss of a coin (52 per cent) in the 24 cases of hepatocellular jaundice and fairly accurate (88 per cent) in the 26 cases of obstructive jaundice.

The results of case analysis to determine the per cent error in total clinical management showed that 5 patients with hepatocellular jaundice were operated upon needlessly and that no patient with a case of obstructive jaundice was denied surgery by diagnostic error. Thus the final accuracy of clinical management was 90 per cent correct. It is important to qualify this statement further by pointing out that this per cent accuracy depended upon the net combination of history, physical examination, additional liver function tests, and in some cases, liver punch biopsy.

Since part of the purpose of this report is to evaluate the use of the intravenous galactose tolerance test, by itself, and in combination with the alkaline phosphatase and thymol turbidity tests, it is of interest to determine the number of cases in which the result of the test, whether positive or negative was of diagnostic significance. If we exclude the cases with impaired renal function (azotemia) we find that 15 out of 20 cases of hepatocellular jaundice had an abnormal galactose test and that 13 out of 19 cases of obstructive jaundice were normal. Thus in approximately 70 per cent of these patients without azotemia the test was of diagnostic significance. Of the 5 patients with hepatocellular jaundice that gave

TABLE I

24 patients with hepatocellular jaundice

Case No.	Age	Diagnosis by History and Physical Exam.	Clinical Management Correct	Final Diagnosis	Intraven. Galactose Tolerance (75′ Resid.)	Serum Alk. Phos. (Bodansky Units)	Serum Thymol Turbidity ($CuSO_4$ Units)	Serum Bilirubin (mg. %)	Non-Protein Nitrogen (mg. %)	Duration of Jaundice	Proof of Diagnosis
Group A (Cases in which laboratory tests were indicative)											
1	75	Obstructive	Yes	Hepatitis	84.4	2.4	6.6	24	34	14 days	Biopsy
2	40	Obstructive	Yes	Hepatitis	43.3	4.6	13.4	21	Urine normal	15 days	Biopsy
3	81	Obstructive	No	Acute yellow atrophy	74.1	7.6	5.3	12.0	Urine normal	11+ days	Surgery
4	43	Obstructive	Yes	Hepatitis	43.1	5.4	14.5	18.0	Urine normal	18 days	Biopsy
5	14	Hepatitis	Yes	Hepatitis	3.3	8.6	17.8	4.2	31	4 days	Biopsy
6	70	Hepatitis	Yes	Cirrhosis	48.8	3.2	15.2	18.0	40	2 weeks	Biopsy
7	39	Cirrhosis	Yes	Cirrhosis	42.2	6.3	8.8	7.2	21	3.5 months	Biopsy
8	29	Obstructive	Yes	Hepatitis	37.7	4.8	19.3	1.8	42	3 weeks	Biopsy
9	59	Cirrhosis	Yes	Cirrhosis	57.3	3.8	28.0	2.5	30	2 months	Biopsy
10	27	Hepatitis	Yes	Cholang. hep.	19.9	6.1	5.9	8.0	Urine normal	11 days	Biopsy
11	36	Hepatitis	Yes	Hepatitis	59.0	5.7	2.2	8.0	36	6 weeks	Biopsy
12	75	Obstructive	No	Cholang. hep.	40.2	14.5	0.6	3.0	48	7 days	Surgery
13	48	Hepatitis	Yes	Hepatitis	95.9	4.4	22.8	4.0	29	4 months	Biopsy
14	45	Cirrhosis	Yes	Cirrhosis	34.8	7.7	19.2	24.0	51	1 year	Biopsy
15	74	Obstructive	Yes	Cirrhosis	46.7	13.8	10.3	10.0	38	6 weeks	Biopsy
16	39	Obstructive	Yes	Cirrhosis	19.1	8.8	12.4	14.2	21	2 weeks	Biopsy
17	75	Hepatocellular	Yes	Cholang. hep.	32.4	17.7	0.8	3.0	40	1 month	Biopsy
18	34	Obstructive	No	Thorazine hep.	17.4	9.3	3.7	7.2	33	2 weeks	Surgery
19	64	Obstructive	Yes	Cirrhosis	52	4.9	3.1	20	32	2 weeks	Autopsy
Group B (Cases in which laboratory tests were not indicative)											
20	41	Cirrhosis	Yes	Cholang. hep.	6.2	8.1	7.0	29.0	24	6 weeks	Biopsy
21	69	Cirrhosis	Yes	Cirrhosis	11.6	4.1	4.5	16.0	29	5 weeks	Autopsy
22	33	Hepatitis	Yes	Cholang. hep.	7.1	15.2	5.0	4.4	25	2½ weeks	Biopsy
23	81	Obstructive	No	Thorazine hep.	1.3	5.8	1.7	28	27	6 weeks	Surgery
24	36	Obstructive	No	Thorazine hep.	19.5	9.1	5.9	12.0	47	4 weeks	Surgery

TABLE II

TABLE II

26 patients with obstructive jaundice

Case No.	Age	Diagnosis by History and Physical Exam.	Clinical Management Correct	Final Diagnosis	Intraven. Galactose Tolerance (75′ Resid.)	Serum Alk. Phos. (Bodansky Units)	Serum Thymol Turbidity ($CuSO_4$ Units)	Serum Bilirubin (mg. %)	Non-Protein Nitrogen (mg. %)	Duration of Jaundice	Proof of Diagnosis
Group A (Cases in which laboratory tests were indicative)											
1	76	Obstructive	Yes	C. D. stone	20.5	20.0	1.8	11.2	32	5 weeks	Surgery
2	44	Hepatitis	Yes	C. D. stone	8.1	6.8	8.7	16.0	Urine normal	2 weeks	Surgery
3	76	Obstructive	Yes	CA pancreas	8.2	15.5	1.7	6.5	36	5 weeks	Surgery
4	76	Obstructive	Yes	CA pancreas	9.7	28.5	1.0	5.0	24	2 weeks	Surgery
5	68	Obstructive	Yes	CA pancreas	30.8	9.3	2.3	34.6	57	10 weeks	Surgery
6	65	Obstructive	Yes	CA pancreas	10.1	27.6	0.6	8.0	25	3 weeks	Surgery
7	72	Obstructive	Yes	CA bile duct	16.2	18.5	1.1	23.5	34	2 months	Surgery
8	27	Obstructive	Yes	C. D. stone	1.7	7.1	0.9	9.6	24	1 week+	Surgery
9	60	Obstructive	Yes	C. D. stone	9.9	22.4	1.7	6.9	Urine normal	2½ months	Surgery
10	76	Hepatitis	Yes	Stricture C. D.	13.0	22.7	0.6	11.5	24	4 weeks	Surgery
11	56	Cirrhosis	Yes	CA pancreas	10.1	9.4	0.6	25.1	Urine normal	1 month	Surgery
12	72	Obstructive	Yes	CA pancreas	12.4	9.5	0.0	10.0	32	2 weeks	Surgery
13	75	Obstructive	Yes	CA pancreas plus cirrhosis (mild)	16.6	25.6	1.1	11.8	27	2 weeks	Autopsy
14	81	Obstructive	Yes	CA pancreas with liver metastases	23.0	14.2	0.6	12.5	54	5 days	Surgery
15	66	Obstructive	Yes	CA pancreas	33.0	29.3	3.3	7.0	87	3 days	Surgery
16	78	Obstructive	Yes	C. D. stone	15.4	14.8	3.0	12.0	54	7 days	Surgery
17	24	Obstructive	Yes	C. D. stone	8.3	4.7	1.4	4.0	Urine normal	5 days	Surgery
18	65	Obstructive	Yes	CA pancreas	4.2	13.4	1.0	10.7	32	27 days	Surgery
19	61	Obstructive	Yes	C. D. stone	2.2	13.4	1.0	5.0	Urine normal	5 days	Surgery
20	80	Obstructive	Yes	Metastatic CA, liver—rectum	31.7	40.6	5.9	20.5	33	4 days	Surgery
21	74	Obstructive	Yes	CA bile duct	30.8	74.7	6.7	25	34.5	3 weeks	Autopsy
22	67	Obstructive	Yes	CA bile duct	45.7	41.0	2.4	40.0	96	6 months	Autopsy
23	15	Obstructive	Yes	C. D. stone	11.7	5.8	1.7	1.6	34	1 month	Surgery
Group B (Cases in which laboratory tests were not indicative)											
24	78	Obstructive	Yes	C. D. stone	26.7	6.0	1.5	15.3	28	6 weeks	Surgery
25	78	Obstructive	Yes	CA bile duct	16.8	12.2	17.4	9.0	41	1 month	Autopsy (refused surgery)
26	69	Obstructive	Yes	C. D. stone	3.1	10.9	12.5	Markedly icteric	48	3 weeks	Surgery

unexpected normal results there was no apparent relation to the duration of jaundice (4 days, 2½ weeks, 5 weeks, 6 weeks and 6 weeks respectively). The diagnoses in these 5 cases included 2 cases of cholangiolytic hepatitis, 1 case of portal cirrhosis, 1 case of acute infectious hepatitis, and 1 case of jaundice due to chlorpromazine. Of the 6 patients with obstructive jaundice and with no evidence of renal impairment that gave unexpected abnormal results again there was no apparent relation to the duration of jaundice which was 4 days, 2 weeks, 3 weeks, 5 weeks, 6 weeks and 2 months respectively. The diagnoses included 2 cases of carcinoma of the bile ducts, 1 case of carcinomatosis and 3 cases of common duct stone 1 of which also had cirrhosis and 1 of which had had recurrent episodes of cholangitis for 20 years. Thus it is reasonable to assume that there was sufficient hepatocellular damage in these patients to give an abnormal galactose result.

Another approach to the evaluation of the galactose test is to compare the diagnostic accuracy of the alkaline phosphatase and thymol turbidity test alone with the diagnostic accuracy of the alkaline phosphatase, thymol turbidity and galactose tolerance test in combination. The galactose test raises the per cent accuracy from 62 to 86 per cent by such an analysis.

DISCUSSION

From the above series of 50 jaundiced patients with histologic evidence of the etiology we have observed that the diagnoses based upon the history and physical examination alone is 70 per cent accurate. The difference between the accuracy of the obstructive group (88 per cent) and the hepatocellular group (52 per cent) is undoubtedly due to the history of pain. The diagnostic accuracy of the intravenous galactose, the serum alkaline phosphatase, and the thymol turbidity test in combination is 84 per cent. The final diagnostic accuracy and management of this series based upon the history, physical examination, additional liver function tests, and in some cases liver punch biopsy was 90 per cent. The 5 patients whose cases were not diagnosed correctly and who underwent unnecessary surgical exploration include 3 cases of jaundice due to chlorpromazine (Thorazine), 1 case of cholangiolytic hepatitis and 1 case of acute yellow atrophy. The galactose test was strongly positive in the last patient with no evidence of renal disease and should have contraindicated a surgical exploration. However, this case occurred early in the course of this study before we had had sufficient experience to trust the galactose tolerance test. Unfortunately, the patient died early in her postoperative course. The trauma of the exploration undoubtedly contributed to or hastened her death. One patient who had jaundice due to chlorpromazine also had an elevated galactose test which should have indicated the correct diagnosis.

In general then we may conclude that the intravenous galactose tolerance test is a useful test of hepatocellular function in the presence of jaundice. Thus it is a valuable adjunct to the laboratory tests in differentiating surgical from nonsurgical jaundice. It is a simple test to perform and an easy laboratory determination with a satisfactory degree of accuracy.

SUMMARY

A series of 50 jaundiced patients with histologic evidence of etiology have been presented. The etiology was hepatocellular in 24 patients and obstructive in 26 patients.

The diagnostic accuracy in the series was 90 per cent correct.

The patients with diagnostic errors include 3 cases of jaundice due to chlorpromazine.

The intravenous galactose tolerance test proved to be a useful test of hepatocellular function in the presence of jaundice.

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REFERENCES

1. Bassett, A. M., Althausen, T. L., and Coltrin, G. C.: New galactose test for differentiation of obstructive from parenchymatous jaundice, Am. J. Digest. Dis. *8:* 432 (Nov.) 1941.
2. Benedict, S. R.: Determination of blood sugar, J. Biol. Chem. *76:* 457 (Feb.) 1928.
3. Bodansky, A.: Notes on determination of serum inorganic phosphatase and serum phosphatase activity, Am. J. Clin. Path. (Tech. Supp.) 7: 51 (Sept.) 1937.
4. Colcher, H., Patek, A. J., Jr., and Kendall, F. E.: Galactose disappearance from blood stream: calculation of galactose removal constant and its application as test for liver function, J. Clin. Investigation *25:* 768 (Sept.) 1946.
5. Dominguez, R., and Pomerene, E.: Kinetics of disappearance of galactose from plasma after rapid intravenous injection, Am. J. Physiol. *141:* 368 (May) 1944.
6. Ducci, H.: Thymol test of Maclagan: standardization and adaptation to Evelyn photoelectric colorimeter, J. Lab. & Clin. Med. *32:* 1266 (Oct.) 1947.
7. Freeman, S.: Comparison of effects of hepatectomy and common bile duct obstruction on serum phosphatase of adult dogs, Am. J. Physiol. *164:* 792 (March) 1951.
8. Gammeltoft, A., and Kjerulf-Jensen, K.: Mechanism of renal excretion of fructose and galactose in rabbit, cat, dog and man, Acta Scandinavica *6:* 368, 1943.
9. Ivy, A. C.: Gastrointestinal tract and associated structures, basic science notes, Army Medical Dept., Research and Graduate School, Vol. *5:* 1949.
10. King, E. J., and Aitkin, R. S.: Intravenous galactose-tolerance test, Lancet *2:* 543 (Nov. 2) 1940.
11. Maclagan, N. F.: Liver function tests in diagnosis of jaundice; review of 200 cases, Brit. Med. J. *2:* 197 (Aug. 9) 1947.
12. Maclagan, N. F., and Bunn, D.: Flocculation tests with electrophoretically separated serum proteins, Biochem. J. *41:* 580, 1947.
13. Maclagan, N. F.: Thymol turbidity test as indicator of liver dysfunction, Brit. J. Exper. Path. *25:* 234, 1944.
14. Malloy, H. T., and Evelyn, K. A.: Determination of bilirubin with photoelectric colorimeter, J. Biol. Chem. *119:* 481 (July) 1937.
15. Meyer, K. A., Steigmann, F., and Popper, H.: Laboratory diagnosis and its pitfalls in management of "surgical jaundice", S. Clin. North America *27:* 37 (Feb.) 1947.
16. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Vol. II: Methods, Baltimore, Maryland, Williams & Wilkins Company, 1932, p. 691.
17. Raymond, A. L., and Blanco, J. G.: Blood sugar determination and separation of sugar with live yeast, J. Biol. Chem. *79:* 649 (Oct.) 1928.
18. Sherlock, S.: Biochemical investigation in liver disease; some correlations with hepatic histology, J. Path. & Bact. *58:* 523 (July) 1946.
19. Wang, C. C., and Grossman, M. I.: Nonexcretion of serum alkaline phosphatase by liver and pancreas of normal dogs, Am. J. Physiol. *156:* 256 (Feb.) 1949.
20. Watson, C. J.: Approach to distinction of medical and surgical jaundice, Minnesota Med. *32:* 973, 1947.
21. Watson, C. J., and Rappaport, E. M.: Comparison of results obtained with hanger cephaline cholesterol flocculation test and Maclagan thymol turbidity test in patients with liver disease, J. Lab. & Clin. Med. *30:* 983 (Dec.) 1945.
22. Zieve, L. H., Hanson, M., and Hill, E.: Studies of liver function tests; derivation of correction allowing use of bromsulfalein test in jaundiced patients, J. Lab. & Clin. Med. *37:* 40 (Jan.) 1951.

ACUTE EMPHYSEMATOUS CHOLECYSTITIS

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Acute emphysematous cholecystitis, gaseous cholecystitis, or pneumocholecystitis is an acute inflammation of the gallbladder associated with local infection by a gas forming organism with the formation of gas in the lumen and/or interstitially in the wall of the gallbladder. The disease is not common, but is very striking and has aroused considerable interest among surgeons and radiologists. A review of the literature by Bell, Brown, and Lenhardt in 1953 records 27 reported cases[1].

As with other types of acute cholecystitis, the disease usually is associated with obstruction of the cystic duct. Infection of the dammed gallbladder contents with a gas forming organism produces an accumulation of gas under pressure. Leakage of the gas through the gallbladder mucosa, usually in the ampullary area, allows interstitial dissection of the gallbladder wall. Controversial opinion is that the gas accumulates in the Rolitansky-Aschoff sinuses[2]. Normal gallbladders from the autopsy table and gallbladders with chronic inflammation from surgery were inflated with air under pressure after the cystic duct had been ligated by Heifetz and Wyloge[2]. Roentgenologic and pathologic examination of these gallbladders showed them to be similar to gallbladders removed for acute pneumocholecystitis.

Radiographically emphysematous cholecystitis presents a characteristic picture. An olive shaped area of radiolucency in the right upper quadrant represents the gallbladder lumen filled with gas. This usually is surrounded by a darker radiolucent margin 0.5 to 1 cm. in thickness representing the gas infiltrated gallbladder wall. Gas is not seen in the biliary duct system as in cholecystoduodenal fistula because of the occluded cystic duct. A correct preoperative diagnosis is made by roentgenologic study of the patient with acute cholecystitis. A flat plate roentgenogram of the abdomen should suffice. Fluid levels have been demonstrated by roentgenograms made in vertical or lateral decubitus position[3].

Because of the rarity of the condition and recurrence following cholecystostomy the following case is of interest.

CASE REPORT

G. W., a 69 year old woman was admitted to the hospital on Feb. 6, 1955 at 6:00 a.m., with the chief complaint of upper abdominal pain. Twelve hours previously the patient had been suddenly stricken with severe cramping upper abdominal pain, radiating around the right costal margin and into the interscapular region. When seen by a physician at her home during the attack, this usually phlegmatic woman was writhing and rolling on the floor with pain. The pain was relieved by 100 mg. of demerol intravenously but recurred

Scientific paper read by title during the Tucson assembly of The Southwestern Surgical Congress, Tucson, Arizona, April 16–18, 1956.

about 4 hours later, and gradually built up to its initial severity. There was no vomiting, but nausea was persistent. A normal bowel movement had occurred about 6 hours prior to the onset of pain and the patient had passed gas per rectum since.

System review was otherwise negative. Diabetes had been diagnosed 6 years previously and was well controlled. A melanoma of the skin of the right suprapubic region had been widely excised along with the right femoral and inguinal lymph nodes 18 months previously. Aside from mild edema of the right leg, well controlled with compression bandage, there had been no symptoms or signs related to this illness.

Examination revealed a 69 year old woman who appeared acutely ill and complained bitterly of abdominal pain. Temperature was 99.4 F., pulse rate 108, and blood pressure 180/100. The abdomen was moderately distended. There was diffuse tenderness over the entire upper half of the abdomen, and exquisite tenderness localized to a small area in the

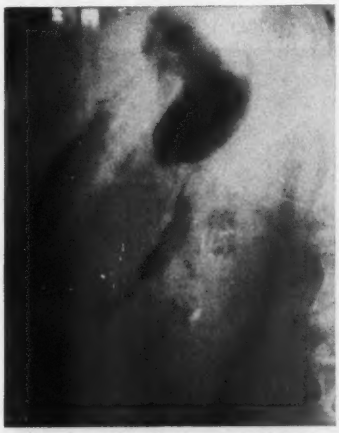

FIG. 1. Feb. 11, 1955. Preoperative flat plate roentgenogram of abdomen showing gallbladder wall outlined by gas.

right subcostal region, associated with a palpable gallbladder. There was no rebound tenderness, and bowel sounds were normal. There were no surgical scars on the abdomen. A surgical scar in the right inguinal region was well healed. Pelvic examination was not done. There was normal colored stool in the rectum.

The white blood cell count was 23,500 per cu. mm. with 90 per cent polymorphonuclears. There was a trace of sugar in otherwise normal urine, and the blood sugar was 223 mg. per cent.

For the following 6 days this patient was treated with gastric aspiration, parenteral fluids, penicillin-streptomycin, terramycin, and insulin as dictated by glycosuria and blood sugar levels. During this period the temperature ranged from 99.2 to 101.2 F. Daily white blood cells showed a gradual decreasing leukocytosis. It was believed that the patient was slowly improving and that gallbladder surgery could be done at a later date as an elective procedure.

A flat plate roentgenogram of the abdomen at the time of admission had been made and an unusual shadow in the right upper quadrant had been accorded no special significance. On the fifth day of the illness cholecystogram was done in the usual fashion with orally administered dye. The unusual shadow again was seen, and the gallbladder was found to be nonfunctioning. Our hospital does not have the services of a full time radiologist, and these films were not interpreted by a radiologist until the day of the cholecystogram. The inter-

pretation was as follows: "There is an unusual gas shadow in the right upper quadrant. It is shell-like, partially following the outline of the kidney shadow but not exactly doing so. The gas shadow is of the order of 5 mm. in thickness at its widest point. On basis of this single view, I would be unable to identify it with certainty, but on basis of subsequent films, it is certain that it represents gas in the gallbladder wall. The other shadows in the abdomen are entirely normal". Two films of the gallbladder area, made on February 11, "Contrast material outlines the gastric mucosa; I presume this represents oral gallbladder dye which has not left the stomach. Again we see the gas shadow in the right upper quadrant of the abdomen. In the oblique view it shifts distinctly anteriorly and can now be identified as the gallbladder. It is now slightly thicker than before. At its point of maximum thickness it is of the order of 6–7 mm. The shadow now surrounds the entire gallbladder. At this time diagnosis of emphysematous cholecystitis can be established".

On the evening of February 11 the patient's condition abruptly became critical with increase in the temperature to 101.8 F., leukocytosis to 21,000, and great increase in severity

FIG. 2. Feb. 17, 1955. Postoperative cholecystocholangiogram. Gallbladder wall shows increased infiltration.

of abdominal pain. A cholecystostomy was performed under local anesthesia through a right subcostal incision. The gallbladder was adherent to the peritoneum of the anterior abdominal wall. It was very large, gray in color, and crepitant to palpation. With a trochar 4 ounces of thin gray malodorous pus was aspirated from the gallbladder. This pus later cultured out B. coli. A large catheter was inserted into the gallbladder and the abdominal wound was closed. Following surgery the patient recovered uneventfully. A cholangiogram performed 6 days postoperatively through the cholecystostomy tube was interpreted as follows: "Three views of the right upper quadrant, made on February 17, are postsurgical, after placement of drainage tube in the gallbladder and after injection of contrast material. The gas shadows of the gallbladder wall are now considerably thicker than at any time before, ranging up to 18 mm. thickness. Contrast material fills the gallbladder, the cystic duct and the common duct. None regurgitates into the hepatic duct system. There is no evidence of stone in the gallbladder itself. In two of the views there is a radiolucent shadow near the fundus of the gallbladder, but in the third view this disappears. I suspect that it represents no more than an air bubble. A small amount of contrast material flows into the adjacent duodenum". The patient was discharged from the hospital on the eleventh postoperative day with the cholecystostomy tube clamped. The tube was removed 6 days later.

Two months later, on April 11, 1955, the patient began to have right upper quadrant

abdominal pain while at work. She was admitted to the hospital and a diagnosis of acute cholecystitis was made. The following day a cholecystectomy was done. Because of the previously reported absence of stones by cholangiogram, a duct exploration was not done. The pathologic report was: "Acute suppurative inflammation superimposed on marked chronic cholecystitis". The convalescence was normal and the patient was discharged on the eleventh postoperative day. She has been well since.

Of the 27 cases reviewed by Bell, Brown, and Lenhardt, cholecystectomy was carried out in 11 patients, cholecystostomy in 7 patients, and 9 patients were treated conservatively[1]. There were 4 deaths in the series. It was the recommendation of these authors that chemotherapy should be combined with surgery in the treatment of acute emphysematous cholecystitis. In consideration of the literature along with the gratifying result in the reported case, surgical drainage or removal of the gallbladder is recommended when the diagnosis of acute emphysematous cholecystitis is made. If the condition of the patient dictates cholecystostomy instead of cholecystectomy, the gallbladder should be removed at a second operation as soon as the patient's condition will permit.

SUMMARY

A case history of a patient with acute emphysematous cholecystitis has been presented. Preoperative diagnosis was made by roentgenogram. Gallstones were not present to account for cystic duct obstruction. Emergency cholecystostomy under local anesthesia brought about dramatic relief of the illness, and secondary cholecystectomy later became necessary. It is recommended that surgical drainage or excision combined with antibiotic therapy be used in patients with this disease.

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REFERENCES

1. Bell, L. G., Brown, R. B., and Lenhardt, H. F.: Acute pneumocholecystitis, Ann. Surg. *138:* 268 (Aug.) 1953.
2. Heifetz, C. J., and Wyloge, E. I.: Effect of distention of gallbladder with air and its relationship to acute pneumocholecystitis, Ann. Surg. *142:* 283 (Aug.) 1955.
3. Retterbush, W. C., Fishman, M. B., Baird, W. A., and Collins, J. I.: Empshysematous cholecystitis, Ann. Surg. *134:* 268 (Aug.) 1951.

SURGICAL TECHNIC

REPAIR OF ESOPHAGEAL HIATAL HERNIA UTILIZING ALLISON'S PRINCIPLE THROUGH AN ABDOMINAL APPROACH

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Esophageal hiatal hernia can be satisfactorily repaired by using an abdominal incision,[2] a thoracic one, or a combined abdominothoracic approach. Each has its advocates, and each gives fairly satisfactory results.

Before thoracic surgery achieved its present maturity, most surgeons thought that abdominal repair was most satisfactory because of freedom from complications associated with thoracotomy. In recent years, thoracic repair has been accepted enthusiastically, particularly because of the findings of Sweet[3], who has pointed out the ease of exposure and dissection with the thoracic incision and the facility of repair with this method.

The swing to the thoracic approach was accelerated by Allison's[1] excellent description of the anatomy of the diaphragmatic crura which form the hiatus. The simplicity of Allison's method, which involves the simple reapposition of the crura with 3 or 4 interrupted silk sutures, has led to its wide use in repair of hiatus hernia. This principle can be utilized with ease from an abdominal incision. This fact has received little, if any, emphasis in the literature.

The advantage of being able to explore the abdomen thoroughly and to treat any coexisting disease makes the abdominal approach of great value. This advantage applies particularly to patients who have proved cholecystic disease or duodenal ulcer, and also to patients where disease other than the hiatal hernia is suspected beforehand but cannot be proved preoperatively.

Although one can readily explore the abdomen through the chest by incising the diaphragm, it is difficult to do cholecystectomy or gastrectomy for ulcer through the chest without enlarging the procedure beyond the indicated scope. Abdominal viscera also can be visualized through an abdominal incision, and one is not limited to palpation alone, which is an important consideration in questionable lesions.

Another consideration is prolonged postoperative pain of the chest wall, which is of significance in about 10 per cent of patients who have thoracotomy, regardless of the way the chest is entered or closed. This pain is obviated with an abdominal incision.

OPERATIVE TECHNIC

An upper midline incision is used, the incision is carried from the xiphoid process to the umbilicus (fig. 1). Occasionally the xiphoid process is divided or removed if an additional inch or 2 of exposure is needed. The advantage of the

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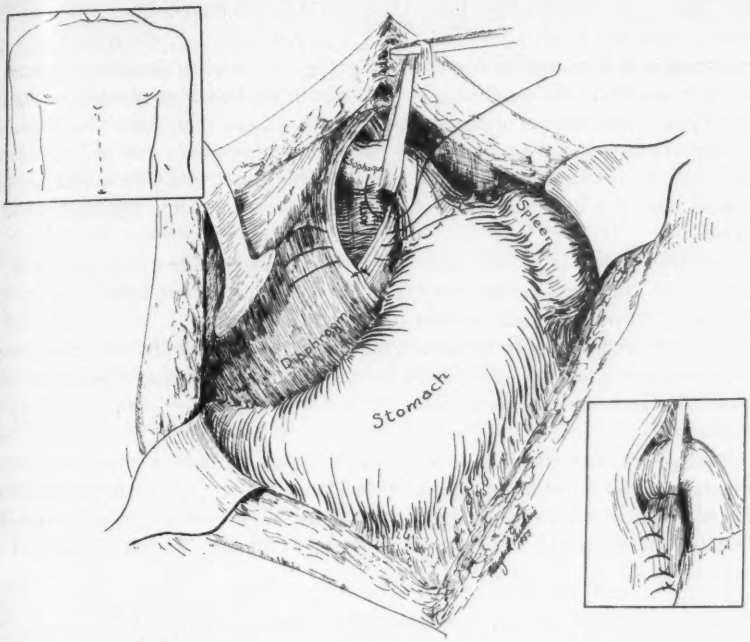

FIG. 1. Repair of hiatus hernia, abdominal approach. The esophagus is retracted upward, and the hiatus closed by suturing the crura together. Upper insert: Incision. Lower insert: Closure of hiatal defect completed.

upper midline incision is that one is working high in the abdomen; thus exposure is better than with the left rectus incision, in which the upper end is limited by the costal margin. In addition, the esophageal hiatus is almost in the midline, and one is operating almost directly over the area of the hiatus.

After the incision is carried into the peritoneal cavity, thorough exploration is done. If there is doubt as to etiology of the patient's complaint or if other disease is found, it might be wise at this point to carry out other procedures before repairing the hiatus.

The left triangular ligament of the liver is divided to expose the hiatus, and the left lobe is folded back on itself (fig. 1). The stomach and other herniated viscera then are gently reduced, and the loose peritoneal tissue overlying the esophagogastric junction is dissected away. If a hernia sac exists, it is thereby removed. A long length of penrose tubing then is passed beneath the lower esophagus and the esophagus retracted directly ventralward and slightly to the patient's left. At the same time, the body of the stomach is retracted dorsally and toward the patient's feet. This maneuver makes taut the fibers of the diaphragmatic crus, even in large defects. One then trims off the loose tissue overlying the muscle making up the crura and is ready to do the repair.

The repair is made using interrupted silk sutures placed about 1 centimeter

apart, exactly as described by Allison,[1] except they are placed from below instead of from above the diaphragm. The hiatus is closed until only the little finger can be inserted into it alongside the esophagus (fig. 1). In some instances it may be necessary to retract the esophagus to the right or to the left as one places sutures in the right or left margin of the hiatus. One also should remember that the aorta lies immediately dorsal to the hiatus and should take care not to puncture it inadvertently. It is not necessary to reinforce the suture line with fascia or other material, and the muscular edges of the crura usually come together without tension.

After the hiatus is closed, the diaphragmaticoesophageal membrane is reattached to the peritoneum overlying the esophagogastric area; the liver is replaced in its normal location and the wound closed.

It has not been necessary to retract the spleen when using the midline incision, and in no patient has injury occurred to the spleen. As the spleen often lies close to the area in which one is working, it is necessary to use care that it is not traumatized.

Although the described method has been used on only a small number of patients and the duration of follow-up has been limited, all patients have been relieved of their symptoms, and to date there have been no recurrences. The following will illustrate the types of cases in which this described method has been useful.

REPORT OF CASES

Case 1. A 43 year old housewife, who was first seen on Jan. 21, 1955, had a chief complaint of hematemesis and pain in the upper portion of the abdomen. She had vomited blood and had tarry stools on several previous occasions. The patient had been studied by other physicians, but none had accurately described the source of bleeding. A diagnosis of duodenal ulcer had been made on one occasion.

Physical examination was negative. Roentgenograms of the upper gastrointestinal tract revealed a hiatal hernia but no evidence of ulcer. The finding of a sliding hiatus hernia was confirmed by esophagoscopy. The decision to do a repair of the hernia through the abdomen was made because of the questionable history of duodenal ulcer and a history of severe asthma. Laparotomy was carried out, and the entire gastrointestinal tract was normal except for the hiatal hernia. Some edema in the esophageal area suggested the presence of esophagitis. The hernia was repaired by using the method described in this paper; the patient recovered and has had no further trouble with bleeding or abdominal pain.

Case 2. A man, 51 years of age, was first seen on Nov. 12, 1951, with a chief complaint of indigestion of 10 years' duration. His indigestion was manifested mainly by belching, abdominal distention, heartburn, nausea, and griping pain in the upper part of the abdomen.

Physical examination was negative, except for tenderness in the epigastrium. Roentgenograms revealed evidence of a small hiatal hernia and a duodenal ulcer. He was placed on a medical program and seemed to do well for about 2 or 3 years.

He returned to the office on May 14, 1953, with exacerbation of his previous symptoms, and a history of melena and hematemesis. The same findings as at his previous study were found, and a stricter medical program was advised.

He returned on Nov. 4, 1954, with a history of almost constant trouble since May 1953, and with little or no benefit from his medical treatment. His pain was in the upper portion of the abdomen, it was worse at night, and it was only partially relieved by antacids. Physical and roentgenogram findings were the same as on previous visits.

Surgical operation was advised and carried out on Dec. 29, 1954. A moderate sized hiatal hernia was repaired and a subtotal gastrectomy was done. The patient had no trouble and was dismissed completely relieved of symptoms.

He has been seen on two occasions since February 1955. He is doing well. Stomach roentgenograms showed no recurrence of the hernia.

SUMMARY

The technic of repairing an esophageal hiatal hernia as described by Allison[1] can easily be applied using an abdominal incision.

Although a thoracic incision is of great merit when one is dealing with an obese patient, the abdominal approach is indicated particularly when other abdominal disease exists or is suspected.

Because the abdominal approach based on Allison's principle facilitates repair, one can often treat other abdominal disease at the same time as hernia repair without adding to the risk of the surgical operation.

Surgeons who treat hiatal hernia should be familiar with all methods of approach and should be able to use the one most practicable for the problem presented.

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REFERENCES

1. Allison, P. R.: Reflux esophagitis, sliding hiatal hernia and anatomy of repair, Surg., Gynec. & Obst. *92:* 419 (April) 1951.
2. Harrington, S. W.: Esophageal hiatal diaphragmatic hernia, Surg., Gynec. & Obst. *100:* 277 (March) 1955.
3. Sweet, R. H.: Repair of hiatus hernia of diaphragm by supradiaphragmatic approach, New England M. Med. *238:* 649 (May 6) 1948.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

A Venture Forward, a History of The American College of Hospital Administrators, compiled under the direction of the history committee by IRA A. KIPNIS. This book may be obtained from headquarters office at 620 North Michigan Avenue, Chicago 11, Ill. $5.00.

Radium Therapy, 2nd. Ed. C. W. WILSON, M.Sc., Ph.D., F.Inst.P., Principal Physicist to the Westminster Hospital, London. Foreword by SIR STANFORD CADE, K.B.E., C.B., D.Sc., F.R.C.S., M.R.C.P., F.F.R., Senior Surgeon, the Westminster Hospital, London, Baltimore, Maryland, The Williams & Wilkins Company. $7.50.

Campbell's Operative Orthopaedics, Volume I and II, edited by J. S. SPEED, M.D., and R. A. KNIGHT, M.D., St. Louis, The C. V. Mosby Company. $40.00.

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